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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
39TH YEAR—ESTABLISHED 1956

- Editorials:
  - Routine Measurement of Work of Breathing: Is It Necessary?
  - Work of Breathing: How To Define and When To Measure?
- A Comparison of Three Methods of Metered Dose Bronchodilator Delivery to a Mechanically Ventilated Adult Lung Model
- Flow-Triggering Does Not Decrease the Work of Breathing and Pressure-Time Product in COPD Patients
- A New Respiratory Monitor That Enables Accurate Measurement of Work of Breathing: A Validation Study
- Unilateral Lung Disease Treated with Patient-Triggered Independent-Lung Ventilation: A Case Report
- The Use of Exogenous Surfactant in Three Pediatric Patients following Gastric Aspiration

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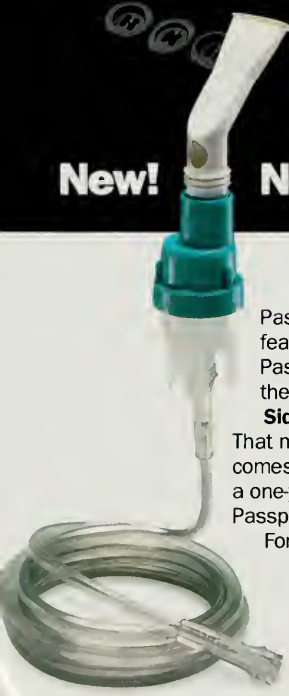

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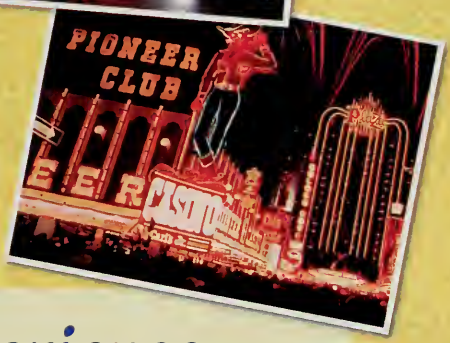

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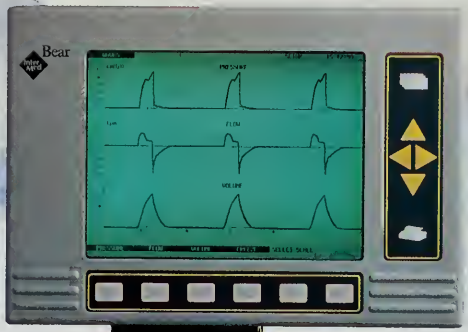

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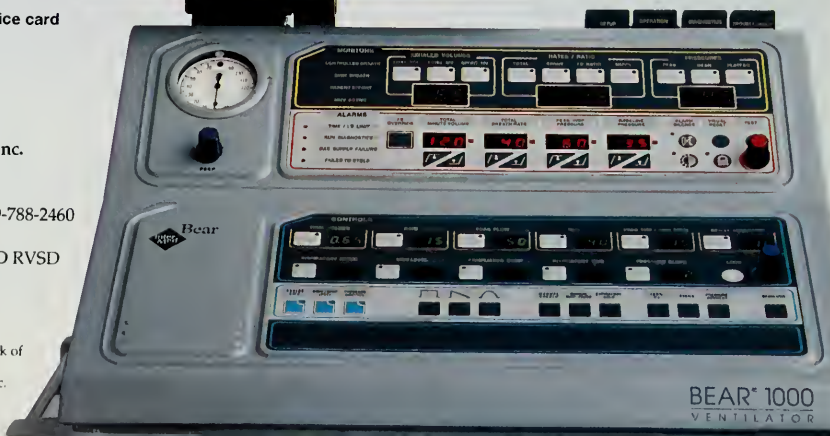

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916 Working group omitted from Static Lung Volumes CPG published in August [Respir Care 1994;39(8):830-836]

916 Citation incorrect for Question 22 of ***CRCE through the Journal*** published in August issue [Respir Care 1994;39(8):842-849]

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Quality Health Care (Contempo 1994)—LA Headrick, D Neuhauser. JAMA 1994;271(21):1711-1712.

Pulmonary and Critical Care Medicine (Contempo 1994)—DR Dantzker, H Steinberg. JAMA 1994;271(21):1709-1710.

Neonatology (Contempo 1994)—P Tarczy-Hornoch, WA Hodson. JAMA 1994;271(21):1682-1684.

Consensus Conference: Practical Applications of Pulmozyme® (report)—BW Ramsey, HL Dorkin for the Consensus Committee. Pediatr Pulmonol 1994;17:404-408.

Why We Need Medical Savings Accounts (Sounding Board)—P Gramm. N Engl J Med 1994;330(24):1752-1753.

Pulmonary-Function Testing (review)—RO Crapo. N Engl J Med 1994;331(1):25-30.

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Outcome Measures for Clinical Trials in Cystic Fibrosis (special article)—BW Ramsey, TF Boat. J Pediatr Med 1994;124(2):177-192.

Clinical Significance of Herpesviral Reactivation in Critically Ill Patients (special issue)—Chest 1994;106(1, Suppl):1S-35S.

Phrenic Nerve Stimulation during Halothane Anesthesia: Effects on Atelectasis—G Hedenstierna, L Tokics, H Lundquist, T Andersson, A Strandberg, B Brismar. Anesthesiology 1994;80(4):751.

BACKGROUND: Atelectasis formation during anesthesia may be due to loss of respiratory muscle tone, in particular that of the diaphragm. This was tested by tensing the diaphragm by phrenic nerve stimulation (PNS) and observing the effect on atelectasis. METHODS: Twelve patients (mean age 48 yr) without preexisting lung disease were studied during halothane anesthesia. PNS was executed with an external electrode on the right side of the neck. Chest dimensions and area of atelectasis were studied by computed tomography of the chest. RESULTS: Right-sided PNS against an occluded airway at functional residual capacity reduced the atelectatic area in the right lung from 5.1 to 3.8 cm^2. The atelectasis was reduced to 1.1 cm^2 after application of positive end-expiratory pressure (PEEP) of 10 cm H_2O and large tidal volumes but increased to 2.5 cm^2 within 1 min after discontinuation of PEEP. Commencement of PNS immediately after PEEP prevented the atelectasis from increasing, the mean area being 0.9 cm^2. In seven patients, in whom the trachea was intubated with a double-lumen endobronchial catheter the atelectatic area was smaller during PNS with an open airway than during positive pressure inflation of the lung with the same volume as inspired during PNS (3.5 and 5.2 cm^2, respectively). CONCLUSIONS: The findings indicate that contracting the diaphragm in the anesthetized subject reduces the size of atelectasis.

***Listeria monocytogenes* and Severe Newborn Respiratory Failure Supported with Extracorporeal Membrane Oxygenation**—RB Hirschl, M Butler, CE Coburn, RH Bartlett, S Baumgart. Arch Pediatr Adolesc Med 1994;148(5):513.

OBJECTIVE: To determine the efficacy of extracorporeal membrane oxygenation (ECMO) in newborn infants with early-onset *Listeria monocytogenes* infection, necrotizing pneumonia, and severe respiratory failure. DESIGN: Patient series. SETTING: ECMO referral centers. PARTICIPANTS: The Extracorporeal Life Support Organization Registry database of patients supported with ECMO between 1975 and 1991. INTERVENTION: ECMO. MEASUREMENT & RESULTS: Nine neonates were identified who were supported with ECMO for severe respiratory failure associated with *L monocytogenes* infection. Microbiologic studies demonstrated *L monocytogenes* organisms in the blood of all infants, and pneumonia was diagnosed by roentgenogram and/or isolation of *L monocytogenes* organisms in tracheobronchial secretions. All infants experienced progressive respiratory deterioration by age 36 hours and were placed on venoarterial bypass by 96 hours, having met institution-based criteria predictive of 80% to 90% mortality. The duration of ECMO for patients with *Listeria* infection (median, 210 hours; range, 137 to 454 hours) was prolonged compared with the duration of ECMO for neonates in all other registry diagnostic categories (median 114 hours; range 1 to 744 hours; N = 5,146, p = 0.035). Six of the nine infants recovered completely. CONCLUSION: These data suggest that ECMO is efficacious in patients with severe respiratory failure secondary to *Listeria* sepsis. Prolonged time on bypass should be expected when *Listeria* sepsis is associated with severe necrotizing pneumonia.

Impact of Antenatal Diagnosis on Survival of Congenital Diaphragmatic Hernia in Minnesota—RH Steinhorn, PK Kriesmer, TP Green, CJ McKay, NR Payne. Arch Pediatr Adolesc Med 1994;148:626.

OBJECTIVE: We characterized the natural history and true mortality of congenital diaphragmatic hernia (CDH) newborn patients by identifying all infants born with this condition in a fixed geographic region over a 2-year period. We examined this population to determine the frequency of intrauterine diagnosis, the outcome of prenatally diagnosed infants, and the impact of deaths in infants with an unsuspected diagnosis (the "hidden mortality") on the overall outcome of this condition. DESIGN: This was a retrospective population survey of all infants born with CDH in Minnesota between June 1988 and June 1990. SETTING: All Minnesota birth and death records were reviewed to identify patients with the diagnosis of CDH. A separate survey of all level 3 intensive care nurseries was conducted and the record of each identified patient was reviewed. Extracorporeal membrane oxygenation was available throughout the study period. MAIN OUTCOME MEASURE: Survival to hospital discharge and short-term morbidity were examined for each patient. RESULTS: Survival was 60% (29/48). Eleven of 19 deaths occurred in patients born prematurely and/or with coexisting major anomalies. Eight percent (4/48) of patients died within the first hour of life prior to diagnosis (hidden mortality). Intrauterine diagnosis of CDH was made in 15 patients. Survival was 60% (9/15) in infants whose con-

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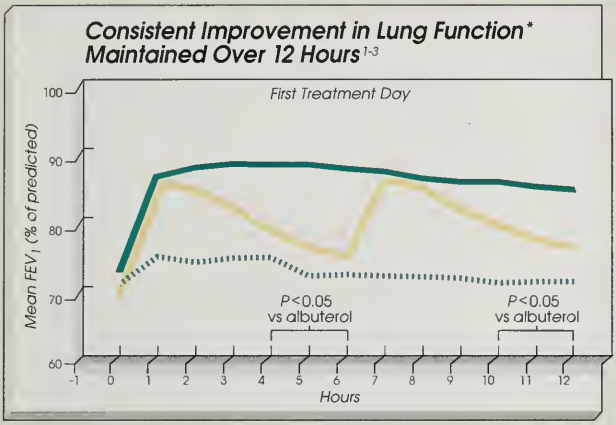

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†*Dosing should be two puffs (42 µg) of SEREVENT Inhalation Aerosol, twice daily, morning and evening, approximately 12 hours apart, for the maintenance treatment of asthma. SEREVENT should not be used for the treatment of acute exacerbations.*

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BRIEF SUMMARY

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CONTRAINDICATIONS: Serevent® Inhalation Aerosol is contraindicated in patients with a history of hypersensitivity to any of the components.

WARNINGS: 1. Not for Use to Treat Acute Symptoms; Watch for Increased Need for Short-Acting $Beta_2$-Agonists: Serevent® Inhalation Aerosol should not be used to relieve acute asthma symptoms. If the patient's short-acting, inhaled $beta_2$-agonist becomes less effective, e.g., the patient needs more inhalations than usual, medical evaluation must be obtained immediately and increasing use of Serevent Inhalation Aerosol in this situation is inappropriate. Serevent Inhalation Aerosol should not be used more frequently than twice daily (morning and evening) at the recommended dose. When prescribing Serevent Inhalation Aerosol, patients must be provided with a short-acting, inhaled $beta_2$-agonist (e.g., albuterol) for treatment of symptoms that occur despite regular twice-daily (morning and evening) use of Serevent.

Asthma may deteriorate acutely over a period of hours or chronically over several days. In this setting, increased use of inhaled, short-acting $beta_2$-agonists is a marker of destabilization of asthma and requires re-evaluation of the patient and consideration of alternative treatment regimens, especially inhaled or systemic corticosteroids. If the patient uses four or more inhalations per day of a short-acting $beta_2$-agonist on a regular basis, or if more than one canister (200 inhalations per canister) is used in an 8-week period, then the patient should see the physician for re-evaluation of treatment.

2. Use With Short-Acting $Beta_2$-Agonists: When patients begin treatment with Serevent Inhalation Aerosol, those who have been taking short-acting, inhaled $beta_2$-agonists on a regular daily basis should be advised to discontinue their regular daily-dosing regimen and should be clearly instructed to use short-acting, inhaled $beta_2$-agonists only for symptomatic relief if they develop asthma symptoms while taking Serevent Inhalation Aerosol (see PRECAUTIONS: Drug Interactions).

3. Serevent Inhalation Aerosol is Not a Substitute for Oral or Inhaled Corticosteroids: Patients must be warned not to stop or reduce corticosteroid therapy without medical advice, even if they feel better when they are being treated with Serevent.

4. Do Not Exceed Recommended Dose: As with other beta-adrenergic aerosols, Serevent Inhalation Aerosol should not be used in excess. Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs. Large doses of inhaled or oral salmeterol (12 to 20 times the recommended dose) have been associated with clinically significant prolongation of the QT_C interval, which has the potential for producing ventricular arrhythmias.

5. Paradoxical Bronchospasm: As with other inhaled asthma medications, paradoxical bronchospasm (which can be life-threatening) has been reported following the use of Serevent Inhalation Aerosol. If it occurs, treatment with Serevent Inhalation Aerosol should be discontinued immediately and alternative therapy instituted.

6. Immediate Hypersensitivity Reactions: Immediate hypersensitivity reactions may occur after administration of Serevent Inhalation Aerosol, as demonstrated by rare cases of urticaria, rash, and bronchospasm.

PRECAUTIONS:
General: 1. Use with Spacer or Other Devices. The safety and effectiveness of Serevent® Inhalation Aerosol when used with a spacer or other devices have not been adequately studied.

2. Cardiovascular and Other Effects: No effect on the cardiovascular system is usually seen after the administration of inhaled salmeterol in recommended doses, but the cardiovascular and central nervous system effects seen with all sympathomimetic drugs (e.g., increased blood pressure, heart rate, excitement) can occur after use of Serevent Inhalation Aerosol and may require discontinuation of the drug. Salmeterol, like all sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension; in patients with convulsive disorders or thyrotoxicosis; and in patients who are unusually responsive to sympathomimetic amines.

As has been described with other beta-adrenergic agonist bronchodilators, clinically significant changes in systolic and/or diastolic blood pressure, pulse rate, and electrocardiograms have been seen infrequently in individual patients in controlled clinical studies with salmeterol.

3. Metabolic Effects: Doses of the related $beta_2$-adrenoceptor agonist albuterol, when administered intravenously, have been reported to aggravate pre-existing diabetes mellitus and ketoacidosis. No effects on glucose have been seen with Serevent Inhalation Aerosol at recommended doses. Administration of $beta_2$-adrenoceptor agonists may cause a decrease in serum potassium, possibly through intracellular shunting, which has the potential to increase the likelihood of arrhythmias. The decrease is usually transient, not requiring supplementation.

Clinically significant changes in blood glucose and/or serum potassium were seen rarely during clinical studies with long-term administration of Serevent Inhalation Aerosol at recommended doses.

Information for Patients: See illustrated Patient's Instructions for Use. **SHAKE WELL BEFORE USING.** Patients should be given the following information:

1. Not for Use to Treat Acute Symptoms: Serevent Inhalation Aerosol is not meant to relieve acute asthmatic symptoms. Acute symptoms should be treated with an inhaled, short-acting bronchodilator that has been prescribed by a physician for symptom relief.

2. Do Not Exceed Recommended Dose: The bronchodilator action of salmeterol usually lasts for at least 12 hours. Therefore it should not be used more often than every 12 hours.

3. Use With Other Medications: While using Serevent Inhalation Aerosol, other inhaled medicines should be taken only as directed by the physician.

4. Use with Short-Acting, Inhaled Beta-Agonists: While using Serevent Inhalation Aerosol, medical attention should be sought immediately if the short-acting bronchodilator treatment becomes less effective for symptom relief, if more inhalations than usual are needed, or if more than the maximum number of inhalations of short-acting bronchodilator treatment prescribed for a 24-hour period are needed. If the patient uses four or more inhalations per day of a short-acting $beta_2$-agonist on a regular basis, or if more than one canister (200 inhalations per canister) is used in an 8-week period, then the patient should see the physician for re-evaluation of treatment.

Patients should be cautioned regarding potential adverse cardiovascular effects, such as palpitations or chest pain, related to the use of additional $beta_2$-agonist.

5. Use of Systemic or Inhaled Steroids: Serevent® (salmeterol xinafoate) Inhalation Aerosol does not replace oral or inhaled corticosteroids; the dosage of these medicines should not be changed and they should not be stopped without consulting the physician, even if the patient feels better.

6. Use for Exercise-Induced Bronchospasm: When using Serevent Inhalation Aerosol to prevent exercise-induced bronchospasm, the dose should be administered at least 30 to 60 minutes before exercise.

Drug Interactions: ***Short-Acting Beta-Agonists:*** In the two 3-month, repetitive-dose clinical trials (n=184), the mean daily need for additional $beta_2$-agonist use was 1 to 1½ inhalations per day, but some patients used more. Eight percent of patients used at least eight inhalations per day at least on one occasion. Six percent used 9 to 12 inhalations at least once. There were 15 patients (8%) who averaged over four inhalations per day. Four of these used an average of 8 to 11 inhalations per day. In these 15 patients there was no observed increase in frequency of cardiovascular adverse events. The safety of concomitant use of more than eight inhalations per day of short-acting $beta_2$-agonists with Serevent Inhalation Aerosol has not been established. In 15 patients who experienced worsening of asthma while receiving Serevent Inhalation Aerosol, nebulized albuterol (one dose in most) led to improvement in FEV_1 and no increase in occurrence of cardiovascular adverse events.

Monoamine Oxidase Inhibitors and Tricyclic Antidepressants: Salmeterol should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants because the action of salmeterol on the vascular system may be potentiated by these agents.

Corticosteroids and Cromoglycate: In clinical trials, inhaled corticosteroids and/or inhaled cromolyn sodium did not alter the safety profile of Serevent Inhalation Aerosol when administered concurrently.

Methylxanthines: The concurrent use of intravenously or orally administered methylxanthines (e.g., aminophylline, theophylline) by patients receiving Serevent Inhalation Aerosol has not been completely evaluated. In one clinical trial, 87 patients receiving Serevent Inhalation Aerosol 42 mcg twice daily concurrently with a theophylline product had adverse event rates similar to those in 71 patients receiving Serevent Inhalation Aerosol without theophylline. Resting heart rates were slightly higher in the patients on theophylline but were little affected by Serevent Inhalation Aerosol therapy.

Carcinogenesis, Mutagenesis, Impairment of Fertility: In an 18-month oral carcinogenicity study in CD-mice, salmeterol xinafoate caused a dose-related increase in the incidence of smooth muscle hyperplasia, cystic glandular hyperplasia, and leiomyomas of the uterus and a dose-related increase in the incidence of cysts in the ovaries. A higher incidence of leiomyosarcomas was not statistically significant; tumor findings were observed at oral doses of 1.4 and 10 mg/kg, which gave 9 and 63 times, respectively, the human exposure based on rodent:human AUC comparisons.

Salmeterol caused a dose-related increase in the incidence of mesovarian leiomyomas and ovarian cysts in Sprague Dawley rats in a 24-month inhalation/oral carcinogenicity study. Tumors were observed in rats receiving doses of 0.68 and 2.58 mg/kg per day (about 55 and 215 times the recommended clinical dose [mg/m^2]). These findings in rodents are similar to those reported previously for other beta-adrenergic agonist drugs. The relevance of these findings to human use is unknown.

No significant effects occurred in mice at 0.2 mg/kg (1.3 times the recommended clinical dose based on comparisons of the AUCs) and in rats at 0.21 mg/kg (15 times the recommended clinical dose on a mg/m^2 basis).

Salmeterol xinafoate produced no detectable or reproducible increases in microbial and mammalian gene mutation *in vitro*. No blastogenic activity occurred *in vitro* in human lymphocytes or *in vivo* in a rat micronucleus test. No effects on fertility were identified in male and female rats treated orally with salmeterol xinafoate at doses up to 2 mg/kg orally (about 160 times the recommended clinical dose on a mg/m^2 basis).

Pregnancy: ***Teratogenic Effects: Pregnancy Category C:*** No significant effects of maternal exposure to oral salmeterol xinafoate occurred in the rat at doses up to the equivalent of about 160 times the recommended clinical dose on a mg/mz basis. Dutch rabbit fetuses exposed to salmeterol xinafoate *in utero* exhibited effects characteristically resulting from beta-adrenoceptor stimulation; these included precocious eyelid openings, cleft palate, sternebral fusion, limb and paw flexures, and delayed ossification of the frontal cranial bones. No significant effects occurred at 0.6 mg/kg given orally (12 times the recommended clinical dose based on comparison of the AUCs).

New Zealand White rabbits were less sensitive since only delayed ossification of the frontal bones was seen at 10 mg/kg given orally (approximately 1,600 times the recommended clinical dose on a mg/m^2 basis). Extensive use of other beta-agonists has provided no evidence that these class effects in animals are relevant to use in humans. There are no adequate and well-controlled studies with Serevent Inhalation Aerosol in pregnant women. Serevent Inhalation Aerosol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Use in Labor and Delivery: There are no well-controlled human studies that have investigated effects of salmeterol on preterm labor or labor at term. Because of the potential for beta-agonist interference with uterine contractility, use of Serevent Inhalation Aerosol during labor should be restricted to those patients in whom the benefits clearly outweigh the risks.

Nursing Mothers: Plasma levels of salmeterol after inhaled therapeutic doses are very low (85 to 200 pg/mL) in humans. In lactating rats dosed with radiolabeled salmeterol, levels of radioactivity were similar in plasma and milk. In rats, concentrations of salmeterol in plasma and milk were similar. The xinafoate moiety is also transferred to milk in rats at concentrations of about half the corresponding level in plasma. However, since there is no experience with use of Serevent Inhalation Aerosol by nursing mothers, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Caution should be exercised when salmeterol xinafoate is administered to a nursing woman.

Pediatric Use: The safety and effectiveness of Serevent Inhalation Aerosol in children younger than 12 years of age have not been established.

Geriatric Use: Of the total number of patients who received Serevent Inhalation Aerosol in all clinical studies, 241 were 65 years and older. Geriatric patients (65 years and older) with reversible obstructive airway disease were evaluated in four well-controlled studies of 3 weeks' to 3 months' duration. Two placebo-controlled, crossover studies evaluated twice-daily dosing with salmeterol for 21 to 28 days in 45 patients. An additional 75 geriatric patients were treated with salmeterol for 3 months in two large parallel-group, multicenter studies. These 120 patients experienced increases in AM and PM peak expiratory flow rate and decreases in diurnal variation in peak expiratory flow rate similar to responses seen in the total populations of the two latter studies. The adverse event type and frequency in geriatric patients were not different from those of the total populations studied.

No apparent differences in the efficacy and safety of Serevent Inhalation Aerosol were observed when geriatric patients were compared with younger patients in clinical trials. As with other $beta_2$-agonists, however, special caution should be observed when using Serevent Inhalation Aerosol in elderly patients who have concomitant cardiovascular disease that could be adversely affected by this class of drug. Based on available data, no adjustment of salmeterol dosage in geriatric patients is warranted.

Serevent® (salmeterol xinafoate) Inhalation Aerosol

ADVERSE REACTIONS: Adverse reactions to salmeterol are similar in nature to reactions to other selective β_2-adrenoceptor agonists, i.e., tachycardia; palpitations; immediate hypersensitivity reactions, including urticaria, rash, bronchospasm (see WARNINGS); headache; tremor; nervousness; and paradoxical bronchospasm (see WARNINGS).

Two multicenter, 12-week, controlled studies have evaluated twice-daily doses of Serevent® (salmeterol xinafoate) Inhalation Aerosol in patients 12 years of age and older with asthma. The following table reports the incidence of adverse events in these two studies.

Adverse Experience Incidence in Two Large 12-Week Clinical Trials*

Adverse Event Type	Percent of Patients		
	Placebo n=187	Serevent 42 mcg b.i.d. n=184	Albuterol 180 mcg q.i.d. n=185
Ear, nose, and throat			
Upper respiratory tract infection	13	14	16*
Nasopharyngitis	12	14	11
Disease of nasal cavity/sinus	4	6	1
Sinus headache	2	4	<1
Gastrointestinal			
Stomachache	0	4	0
Neurological			
Headache	23	28	27
Tremor	2	4	3
Respiratory			
Cough	6	7	3
Lower respiratory infection	2	4	2

* The only adverse experience classified as serious was one case of upper respiratory tract infection in a patient treated with albuterol.

The table above includes all events (whether considered drug related or nondrug related by the investigator) that occurred at a rate of over 3% in the Serevent Inhalation Aerosol treatment group and were more common in the Serevent Inhalation Aerosol group than in the placebo group.

Pharyngitis, allergic rhinitis, dizziness/giddiness, and influenza occurred at 3% or more but were equally common on placebo. Other events occurring in the Serevent Inhalation Aerosol treatment group at a frequency of 1% to 3% were as follows:

Cardiovascular: Tachycardia, palpitations.
Ear, Nose, and Throat: Rhinitis, laryngitis.
Gastrointestinal: Nausea, viral gastroenteritis, nausea and vomiting, diarrhea, abdominal pain.
Hypersensitivity: Urticaria.
Mouth and Teeth: Dental pain.
Musculoskeletal: Pain in joint, back pain, muscle cramp/contraction, myalgia/myositis, muscular soreness.
Neurological: Nervousness, malaise/fatigue.
Respiratory: Tracheitis/bronchitis.
Skin: Rash/skin eruption.
Urogenital: Dysmenorrhea

In small dose-response studies, tremor, nervousness, and palpitations appeared to be dose related

OVERDOSAGE: Overdosage with salmeterol may be expected to result in exaggeration of the pharmacologic adverse effects associated with beta-adrenoceptor agonists, including tachycardia and/or arrhythmia, tremor, headache, and muscle cramps. Overdosage with salmeterol can lead to clinically significant prolongation of the QT_c interval, which can produce ventricular arrhythmias. Other signs of overdosage may include hypokalemia and hyperglycemia.

In these cases, therapy with Serevent® Inhalation Aerosol and all beta-adrenergic–stimulant drugs should be stopped, supportive therapy provided, and judicious use of a beta-adrenergic blocking agent should be considered, bearing in mind the possibility that such agents can produce bronchospasm. Cardiac monitoring is recommended in cases of overdosage.

As with all sympathomimetic pressurized aerosol medications, cardiac arrest and even death may be associated with abuse of Serevent Inhalation Aerosol.

Rats and dogs survived the maximum practicable inhalation doses of salmeterol of 2.9 and 0.7 mg/kg, respectively. The maximum nonlethal oral doses in mice and rats were approximately 150 mg/kg and >1,000 mg/kg, respectively

Dialysis is not appropriate treatment for overdosage of Serevent Inhalation Aerosol.

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RL-096

References:

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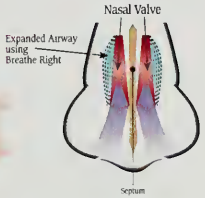

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ditions were diagnosed in utero, a rate identical to that for infants whose conditions were diagnosed in the postnatal period (61% [20/33]). There was no relationship between age at fetal diagnosis and mortality. CONCLUSIONS: The hidden mortality of CDH was low. Almost half of the total mortality for CDH was associated with coexisting, additional anomalies. Patients who were not offered extracorporeal membrane oxygenation owing to prematurity, other major anomalies, or birth at a center that did not offer extracorporeal membrane oxygenation accounted for 84% (16/19) of deaths. These data will be useful for determining the impact of new therapeutic strategies on the mortality of CDH.

Lung Structure and Function in Different Stages of Severe Adult Respiratory Distress Syndrome—L Gattinoni, M Bombino, P Pelosi, A Lissoni, A Pesenti, R Fumagalli, M Tagliabue. JAMA 1994;271:1772.

OBJECTIVE: To assess the clinical consequences of duration of adult respiratory distress syndrome (ARDS) on lung structure and function. DESIGN: Retrospective analysis. SETTING: A university hospital referral center for extracorporeal support. PATIENTS: A total of 84 patients with severe ARDS (Murray score > 2.5) recruited from 48 intensive care units (1979 to 1992), who suffered ARDS and underwent mechanical ventilation for up to 1 week (37 patients with early ARDS), between 1 and 2 weeks (24 patients with intermediate ARDS), or more than 2 weeks (23 patients with late ARDS) and subsequently underwent extracorporeal support. MAIN OUTCOME MEASURES: Before beginning extracorporeal support, we measured gas exchange, pulmonary mechanics, hemodynamics, oxygen transport and delivery, incidence of barotrauma (presence of one or more thoracic tubes for pneumothorax drainage), and organ dysfunctions. In a subgroup of 16 patients, we studied lung structure by computed tomographic scan, scoring the densities and quantifying the emphysemalike lesions (bullae). RESULTS: Late ARDS showed lower respiratory compliance, higher dead space, higher P_{aCO_2}, lower venous admixture, and lower positive end-expiratory pressure requirement compared with early ARDS ($p < 0.01$). The incidence of pneumothorax (48.8% of the entire population) was significantly ($p < 0.01$) higher in late ARDS (87%) vs intermediate ARDS (46%) and early ARDS (30%). The mortality of patients with pneumothorax (66%) was significantly ($p < 0.01$) higher compared with patients without pneumo-thorax (46%). The number of bullae per lung was significantly higher in late ARDS vs intermediate and early ARDS (mean [SD], 8 [4], 4.3 [5], and 1.9 [3.9], respectively; $p < 0.01$), and they were preferentially distributed in the dependent lung regions. The number of bullae per lung was significantly higher in patients with pneumothorax vs those without pneumothorax (mean [SD], 13.6 [9.8] vs 1.4 [2.1], F = 0.007). The mean (SD) duration of ARDS in patients with pneumothorax was 15.3 (10.0) days vs 7.0 (6.6) days in those without pneumothorax ($p = 0.0001$). No differences within the three groups were found in computed tomographic scan densities, hemodynamics, and number of organ system dysfunctions. CONCLUSIONS: The lung structure and function changes markedly with ARDS duration, and the late stages may be described as restrictive lung disease with superimposed emphysemalike lesions. Presence of pneumothorax affects survival and appears to be related to the lung structural changes occurring with time.

Epidemiology of Tuberculosis in San Francisco—PM Small, PC Hopewell, SP Singh, APaz, J Parsonnet, DC Ruston, et al. N Engl J Med 1994;330(24):1703.

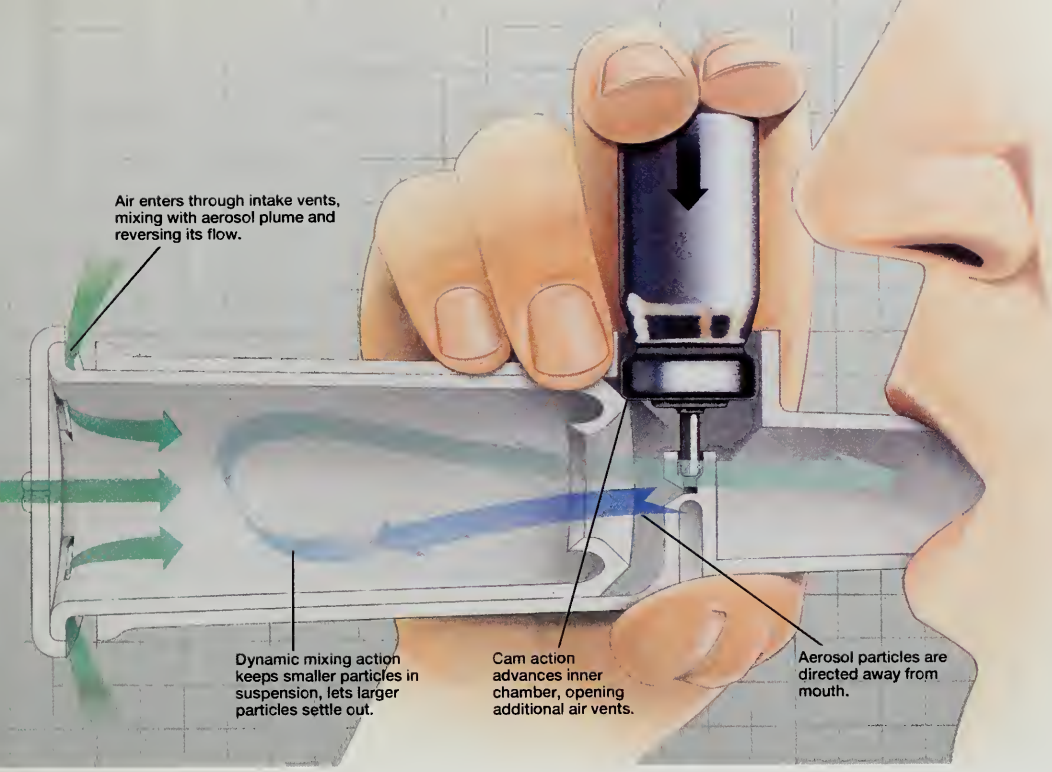
BACKGROUND: The epidemiology of tuberculosis in urban populations is changing. Combining conventional epidemiologic techniques with DNA fingerprinting of *Mycobacterium tuberculosis* can improve the understanding of how tuberculosis is transmitted. METHODS: We used restriction-fragment-length polymorphism (RFLP) analysis to study *M tuberculosis* isolates from all patients reported to the tuberculosis registry in San Francisco during 1991 and 1992. These results were interpreted along with clinical, demographic, and epidemiologic data. Patients infected with the same strains were identified according to their RFLP patterns, and patients with identical patterns were grouped in clusters. Risk factors for being in a cluster were analyzed. RESULTS: Of 473 patients studied, 191 appeared to have active tuberculosis as a result of recent infection. Tracing of patients' contacts with the use of conventional methods identified links among only 10% of these patients. DNA fingerprinting, however, identified 44 clusters, 20 of which consisted of only 2 persons and the largest of which consisted of 30 persons. In patients under 60 years of age, Hispanic ethnicity (odds ratio, 3.3; $p = 0.02$), black race (odds ratio, 2.3; $p = 0.02$), birth in the United States (odds ratio, 5.8; $p < 0.001$), and a diagnosis of the acquired immunodeficiency syndrome (odds ratio, 1.8; $p = 0.04$) were independently associated with being in a cluster. Further study of patients in clusters confirmed that poorly compliant patients with infectious tuberculosis have a substantial adverse effect on the control of this disease. CONCLUSIONS: Despite an efficient tuberculosis-control program, nearly a third of new cases of tuberculosis in San Francisco are the result of recent infection. Few of these instances of transmission are identified by conventional contact tracing.

Transmission of Tuberculosis in New York City: An Analysis by DNA Fingerprinting and Conventiunal Epidemiologic Methods—D Alland, GE Kalkut, AR Moss, RA Mcadam, JA Hahn, W Bosworth, et al. N Engl J Med 1994;330(24):1710.

BACKGROUND: The incidence of tuberculosis and drug resistance is increasing in the United States, but it is not clear how much of the increase is due to reactivation of latent infection and how much to recent transmission. METHODS: We performed DNA fingerprinting using restriction-fragment-length polymorphism (RFLP) analysis of at least one isolate from every patient with confirmed tuberculosis at a major hospital in the Bronx, New York, from December 1, 1989, through December 31, 1992. Medical records and census-tract data were reviewed for relevant clinical, social, and demographic data. RESULT: Of 130 patients with tuberculosis, 104 adults (80%) had complete medical records and isolates whose DNA fingerprints could be evaluated. Isolates from 65 patients (62.5%) had unique RFLP patterns, whereas isolates from 39 patients (37.5%) had RFLP patterns that were identical to those of an isolate from at least 1 other study patient; the isolates in the latter group were classified into 12 clusters. Patients whose isolates were included in one of the clusters were inferred to have recently transmitted disease. Independent risk factors for having a clustered isolate included seropositivity for the human immunodeficiency virus (HIV) (odds ratio for Hispanic patients, 4.31; $p = 0.02$; for non-Hispanic patients, 3.12; $p = 0.07$), Hispanic ethnicity combined with HIV seronegativity (odds ratio, 5.13; $p = 0.05$), infection wlth drug-resistant tuberculosis (odds ratio, 4.52; $p = 0.005$), and younger age (odds ratio, 1.59; $p = 0.02$). Residence in sections of the Bronx with a median household income below $20,000 was also associated with having a clustered isolate (odds ratio, 3.22; $p = 0.04$). CONCLUSIONS: In the inner-city community we studied, recently transmitted tuberculosis accounts for approximately 40% of the incident cases and almost two thirds of drug-resistant cases. Recent transmission of tuberculosis, and not only reactivation of latent disease, contributes substantially to the increase in tuberculosis.

Education Attainment Level of Caregivers Versus Readability Level of Written Instructions in a Pediatric Emergency Department—D Chacon, N Kissoon, S Rich. Pediatr Emerg Care 1994;10(3):144.

OBJECTIVES: (1) to determine the education attainment level (EAL) of parents attending the pediatric emergency department (PED); (2) to assess the readability level (RL) of the written instructions available to these parents; and (3) to revise these instruction forms at a level of comprehension based on the EAL of parents.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We used a questionnaire survey of EAL and RL assessment using the SMOG and FOG readability formulas. We then revised the written instructions and used parents in the PED to pretest them. The setting was a PED in a tertiary care hospital (Children's Hospital of Western Ontario). The participants were 1034 parents of children in the PED between 8 AM and 11 PM over a 6-month period. Parents were chosen at 30-minute intervals on randomly selected weekdays and weekends. A total of 1022 completed the study. Seven forms commonly used in the PED were assessed for RL. Seven forms were rewritten at grade 6 to 7 RL, and each was pretested in 21 to 24 parents or guardians in the PED using a standardized questionnaire with open-ended questions. Ninety percent of respondents spoke English, and 85% had English as their first language. Forty-nine percent of parents had a grade 13 or lower EAL. This included the following EALs: < grade 12 EAL, 23%; < grade 10 EAL, 15%; and < grade 8 EAL, 4%. Of the seven forms tested, five were written at a college RL. Pretesting of revised forms elicited the following responses from parents: easy to understand (100%), understood everything (96-100%), worth remembering (77-96%), liked the form (67-100%), found it informative (52-85%), and thought it was applicable to all people (82-100%). They most frequently commented that the forms were easy to read and easy to understand. Our study showed that discharge instructions are written at a RL at which approximately 50% of the population served may not understand. This is supported by available data suggesting that forms should be written at a grade 6 or lower RL. Properly designed forms that are written at the grade 6 to 7 level were easy to understand and were associated with parental satisfaction. Design of these forms should not only involve medical personnel but also readability consultants and parents or caregivers.

Survival and Long-Term Follow-Up of Tracheostomized Patients with COPD Treated by Home Mechanical Ventilation—JF Muir, C Girault, J Cardinaud, JM Polu, and the French Cooperative Study Group. Chest 1994;106:201.

To define more clearly the value of home mechanical ventilation by tracheostomy (HMVT) in patients with advanced COPD, a retrospective French multicenter study group analyzed the prognostic factors and long-term survival of 259 patients with severe COPD, who were tracheostomized for at least 1 year. Seventy-eight percent of the patients died by the end of the observation period. The actuarial survival rate for the overall study population was, therefore, 70% at 2 years, 44% at 5 years, and 20% at 10 years. These results appear to be better than those of the major published series and compare to the prognosis of COPD patients treated by long-term oxygen therapy (LTO) 15 hr/24 hr. The parameters most closely correlated with a survival for more than 5 years were age < 65 years, use of an uncuffed cannula, and a P_{aO_2} > 55 mm Hg in room air during the 3 months after tracheostomy ($p < 0.01$). This study, therefore, confirmed the feasibility of HMVT in COPD and should lead to a review of the place of permanent tracheostomy in the long-term prognosis of severe COPD patients.

Antioxidant Status in Asthma—CV Powell, AA Nash, HJ Powers, RA Primhak. Pediatr Pulmonol 1994;18:34.

Reduced levels of glutathione peroxidase (GSH-Px) have been observed in adults with asthma. This study examines the antioxidant status in children with asthma compared with a control group in a cross-sectional analysis. Red blood cell GSH-Px, superoxide dismutase (SOD), and plasma concentrations of retinol, vitamin C, alpha tocopherol, and cholesterol were measured in 37 subjects (26 males) with stable controlled asthma. Thirty-five subjects (20 males) without eczema, hayfever, or recurrent respiratory symptoms were used as a control group. Children with asthma had significantly reduced red blood cell GSH-Px activity compared with controls [median (inter-quartile range) for asthma group, 10.25 (9.25-11.91); for control group, 11.75 (10.34–12.26) IU/g Hb; $p = 0.006$]. There were no significant differences in activity of SOD or vitamin C, retinol, or alpha tocopherol/cholesterol ratio. The reduction in GSH-Px activity may have therapeutic and etiological implications for asthma. The effects of disease activity and treatment on antioxidant status needs for further study.

Oral-Nasal Continuous Positive Airway Pressure as a Treatment for Obstructive Sleep Apnea—GL Prosise, RB Berry. Chest 1994;106:180.

The effect of continuous positive airway pressure (CPAP) applied via a mask covering the nose and mouth (oral-nasal CPAP = ONCPAP) on obstructive sleep apnea (OSA) was studied in ten male patients with a mean (± SD) age of 48.1 ± 11.1 years who could not tolerate nasal CPAP (NCPAP) due to nasal congestion. Using ONCPAP at pressures of 11.0 ± 4.5 cm H_2O, the apnea-hypopnea index was reduced from 58.3 ± 22.3 (baseline night) to 5.2 ± 1.6 events per hour (ONCPAP night) ($p < 0.001$). Five of these patients were studied on a subsequent night with a dual chamber mask allowing separate measurement of nasal and oral flow. All patients had combined oral and nasal flow at times during the night, but the fraction of time spent with this breathing pattern was lower during sleep than wakefulness. In a separate study, we compared the effects of a therapeutic level of CPAP pressure (12.8 ± 2.5 cm H_2O) applied through a nasal mask (NCPAP) and ONCPAP in a different group of patients (mean age 60 ± 14.6 years) with moderate to severe OSA using NCPAP on a long-term basis. The apnea-hypopnea indexes on NCPAP nights (7.2±3.5) and ONCPAP nights (7.6 ± 4.9 events per hour of sleep) were very similar. We conclude that ONCPAP may be a reasonable treatment alternative in patients who cannot tolerate NCPAP due to nasal congestion and that the pressure required to maintain upper airway patency may be similar to the level required using NCPAP.

Influence of Positive End-Expiratory Pressure on Right and Left Ventricular Performance Assessed by Doppler Two-Dimensional Echocardiography—G Huemer, N Kolev, A Kurz, M Zimpfer. Chest 1994;106:67.

The advent of modern Doppler two-dimensional ultrasound technology has overcome the need of invasive measurements of several important cardiac parameters. It allows estimation of preload, contractility, and afterload. Positive end-expiratory pressure (PEEP) is associated with a reduction in cardiac output. The responsible mechanisms are controversial. To evaluate the cardiovascular responses to PEEP, we employed different Doppler hemodynamic indices for the first time, combined with conventional two-dimensional echocardiography. Twenty-one healthy, young, and unsedated volunteers were admitted to the study. Under spontaneous respiration, PEEP level was increased stepwise (0, 5, 7.5, 10, 12.5 cm H_2O). At each PEEP level, the following right and left ventricular parameters were assessed with Doppler two-dimensional echocardiography: *two-dimensional variables:* end-diastolic volume indices (EDVI), ejection fraction (EF), and left ventricular afterload—LaPlace relation (combined with cuff systolic pressure); *Doppler variables:* cardiac index (CI) (combined with two-dimensional measure of valve area), maximum velocity (Vmax), time velocity integral (TVI), acceleration time (AT), deceleration time (DT), deceleration rate (DR), ratio of early to atrial peak (E/A), ratio of isovolumic contraction time to ejection time (IVCT/ ET), and maximum blood acceleration (dv/dt) in aorta and main pulmonary artery. Increasing PEEP resulted in a proportional decrease in biventricular EDVI. Moreover, PEEP application is also causing a drop of CI, which is determined from a decrease in Vmax and TVI, while EF, IVCT/ET, dv/dt, Doppler transatrioventricular parameters, and afterload stay in normal ranges. Employing Doppler hemodynamic indices for the first time in this study setting clearly supports data that the drop in EDVI and CI during PEEP is caused by reduction in ventricular filling due to decreased venous return. Using the Doppler parameters IVCT/ET and dv/dt, changes in myocardial contractility, as well as changes in afterload (LaPlace relation), can be ruled out.

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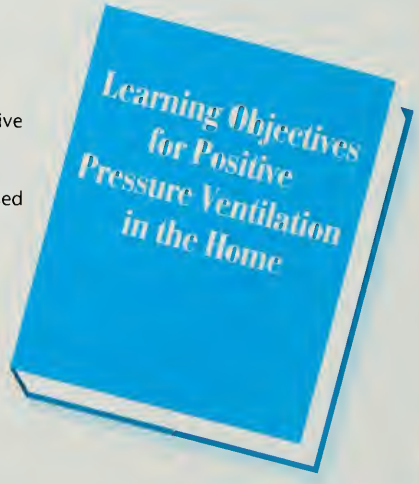

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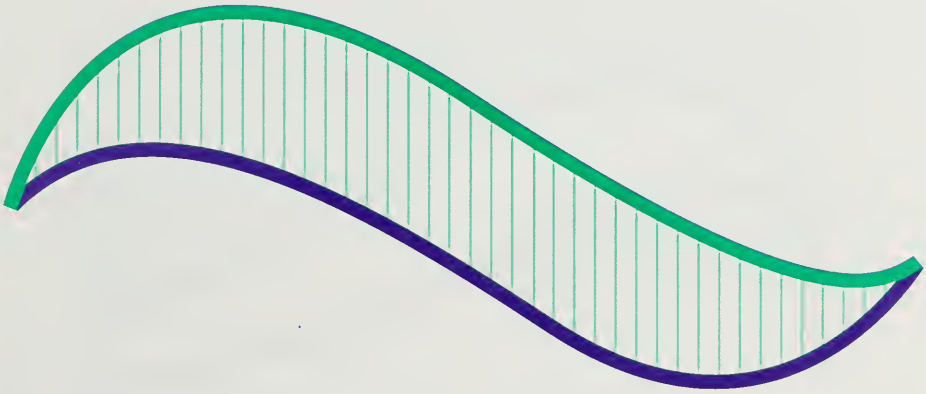
A NEW SOLUTION IN NEBULIZATION



Working Together for Added FEV_1 in COPD

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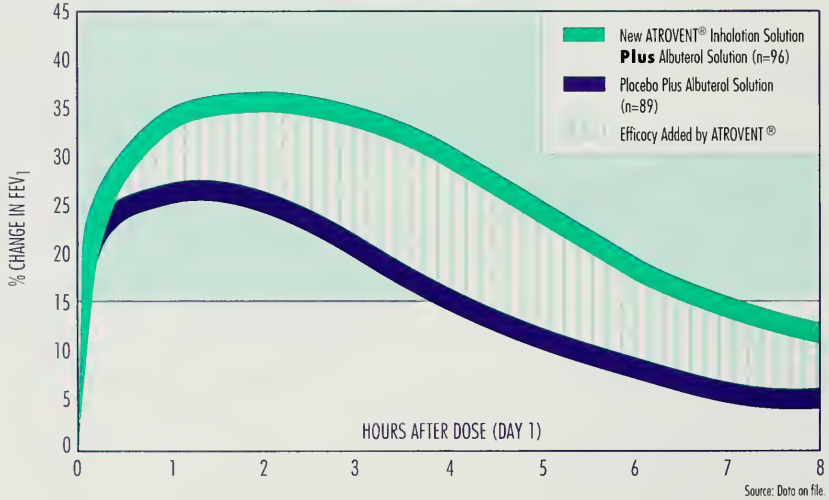
ATROVENT®

(ipratropium bromide) 500 mcg unit dose 0.02%

INHALATION SOLUTION

Significantly raises the efficacy curve

In combination with albuterol



Source: Data on file.

In patients with COPD, ATROVENT Inhalation Solution plus albuterol solution produced significantly greater improvement in FEV_1 than albuterol alone.[1]*

ATROVENT Inhalation Solution is a bronchodilator for the maintenance treatment of bronchospasm in COPD.

* Multicenter, randomized, double-blind, parallel trial conducted in 195 patients with COPD in whom pretreatment mean FEV_1 = 39% of predicted normal

Please see brief summary of Prescribing Information on last page of this advertisement.

. . .without lowering the safety profile

The combination of ATROVENT Inhalation Solution and albuterol solution produced no clinically significant increase in side effects compared with albuterol alone.[2]

ATROVENT Inhalation Solution has a low incidence of systemic anticholinergic side effects.[3]

The use of ATROVENT Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation.

Combination of ATROVENT and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation.

ATROVENT should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy or bladder-neck obstruction.

Please see Prescribing Information for all adverse events, regardless of drug relationship, reported in the 12-week controlled trials of ATROVENT Inhalation Solution.

NEW

ATROVENT®

(ipratropium bromide) 500 mcg unit dose 0.02%

INHALATION SOLUTION

Added efficacy without added side effects*

*When used in combination with a beta-agonist solution, ATROVENT does not significantly increase side effects associated with the beta agonist alone.

NEW

ATROVENT®
(ipratropium bromide) 500 mcg unit dose 0.02%
INHALATION SOLUTION

ADDED EFFICACY WITHOUT ADDED SIDE EFFECTS*

Atrovent®
(ipratropium bromide)
Inhalation Solution

Brief Summary of Prescribing Information

CONTRAINDICATIONS Atrovent® (ipratropium bromide) is contraindicated in known or suspected cases of hypersensitivity to ipratropium bromide, or to atropine and its derivatives

WARNINGS The use of Atrovent® (ipratropium bromide) Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied Drugs with faster onset of action may be preferable as initial therapy in this situation Combination of Atrovent and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation

PRECAUTIONS General Atrovent® (ipratropium bromide) should be used with caution in patients with narrow angle glaucoma, prostatic hypertrophy or bladder-neck obstruction

Information for Patients Patients should be advised that temporary blurring of vision, precipitation or worsening of narrow-angle glaucoma or eye pain may result if the solution comes into direct contact with the eyes Use of a nebulizer with mouthpiece rather than face mask may be preferable, to reduce the likelihood of the nebulizer solution reaching the eyes Patients should be advised that Atrovent Inhalation Solution can be mixed in the nebulizer with albuterol if used within one hour Compatibility data are not currently available with other drugs Patients should be reminded that Atrovent Inhalation Solution should be used consistently as prescribed throughout the course of therapy

Drug Interactions Atrovent has been shown to be a safe and effective bronchodilator when used in conjunction with beta adrenergic bronchodilators Atrovent has also been used with other pulmonary medications, including methylxanthines and corticosteroids, without adverse drug interactions

Carcinogenesis, Mutagenesis, Impairment of Fertility Two-year oral carcinogenicity studies in rats and mice have revealed no carcinogenic potential at dietary doses up to 6 mg/kg/day of Atrovent Results of various mutagenicity studies (Ames test, mouse dominant lethal test, mouse micronucleus test and chromosome aberration of bone marrow in Chinese hamsters) were negative Fertility of male or female rats at oral doses up to 50 mg/kg/day was unaffected by Atrovent administration At doses above 90 mg/kg, increased resorption and decreased conception rates were observed

Pregnancy *TERATOGENIC EFFECTS Pregnancy Category B* Oral reproduction studies performed in mice, rats and rabbits at doses of 10, 100, and 125 mg/kg respectively, and inhalation reproduction studies in rats and rabbits at doses of 1 5 and 1 8 mg/kg (or approximately 38 and 45 times the recommended human daily dose) respectively, have demonstrated no evidence of teratogenic effects as a result of Atrovent However, no adequate or well-controlled studies have been conducted in pregnant women Because animal reproduction studies are not always predictive of human response, Atrovent should be used during pregnancy only if clearly needed

Nursing Mothers It is not known whether Atrovent is excreted in human milk Although lipid-insoluble quaternary bases pass into breast milk, it is unlikely that Atrovent® (ipratropium bromide) would reach the infant to a significant extent, especially when taken by inhalation, since Atrovent is not well absorbed systemically after inhalation or oral administration However, because many drugs are excreted in human milk caution should be exercised when Atrovent is administered to a nursing woman

Pediatric Use Safety and effectiveness in children below the age of 12 have not been established

ADVERSE REACTIONS Adverse reaction information concerning Atrovent® (ipratropium bromide) Inhalation Solution is derived from 12-week active-controlled clinical trials Additional information is derived from foreign post-marketing experience and the published literature

All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials appear in the table below

All Adverse Events, from Double-blind, Parallel, 12-week Studies of Patients with COPD*

	PERCENT OF PATIENTS				
	Atrovent® (500 mcg t i d) n=219	Alupent® (15 mg t i d) n=212	Atrovent®/Alupent® (500 mcg t i d/ 15 mg t i d) n=108	Albuterol (2 5 mg t i d) n=205	Atrovent®/Albuterol (500 mcg t i d/ 2 5 mg t i d) n=100
Body as a Whole-General Disorders					
Headache	6 4	5 2	6 5	6 3	9 0
Pain	4 1	3 3	0 9	2 9	5 0
Influenza-like symptoms	3 7	4 7	6 5	0 5	1 0
Back pain	3 2	1 9	1 9	2 4	0 0
Chest pain	3 2	4 2	5 6	2 0	1 0
Cardiovascular Disorders					
Hypertension/ Hypertension Aggravated	0 9	1 9	0 9	1 5	4 0
Central & Peripheral Nervous System					
Dizziness	2 3	3 3	1 9	3 9	4 0
Insomnia	0 9	0 5	4 6	1 0	1 0
Tremor	0 9	7 1	8 3	1 0	0 0
Nervousness	0 5	4 7	6 5	1 0	1 0
Gastrointestinal System Disorders					
Mouth Dryness	3 2	0 0	1 9	2 0	3 0
Nausea	4 1	3 8	1 9	2 9	2 0
Constipation	0 9	0 0	3 7	1 0	1 0
Musculo-skeletal System Disorders					
Arthritis	0 9	1 4	0 9	0 5	3 0
Respiratory System Disorders (Lower)					
Coughing	4 6	8 0	6 5	5 4	6 0
Dyspnea	9 6	13 2	16 7	12 7	9 0
Bronchitis	14 6	24 5	15 7	16 6	20 0
Bronchospasm	2 3	2 8	4 6	5 4	5 0
Sputum Increased	1 4	1 4	4 6	3 4	0 0
Respiratory Disorder	0 0	6 1	6 5	2 0	4 0
Respiratory System Disorders (Upper)					
Upper Respiratory Tract Infection	13 2	11 3	9 3	12 2	16 0
Pharyngitis	3 7	4 2	5 6	2 9	4 0
Rhinitis	2 3	4 2	1 9	2 4	0 0
Sinusitis	2 3	2 8	0 9	5 4	4 0

*All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials

Additional adverse reactions reported in less than three percent of the patients treated with Atrovent include tachycardia, palpitations, eye pain, urinary retention, urinary tract infection and urticaria A single case of anaphylaxis thought to be possibly related to Atrovent has been reported Cases of precipitation or worsening of narrow-angle glaucoma and acute eye pain have been reported

Lower respiratory adverse reactions (bronchitis, dyspnea and bronchospasm) were the most common events leading to discontinuation of Atrovent therapy in the 12-week trials Headache, mouth dryness and aggravation of COPD symptoms are more common when the total daily dose of Atrovent equals or exceeds 2,000 mcg

HOW SUPPLIED Atrovent® (ipratropium bromide) Inhalation Solution Unit-Dose Vial is supplied as a 0 02% clear, colorless solution containing 2 5 ml with 25 vials per foil pouch (NDC 0597-0080-62) Each vial is made from a low density polyethylene (LDPE) resin

Consult package insert before prescribing.

AS-BS-9/93

* When used in combination with a beta-agonist solution, ATROVENT does not significantly increase side effects associated with the beta agonist alone

References: 1. Levin DC, Little KS, Laughlin KR, Galbraith JM. Ipratropium bromide solution in COPD augments extent and duration of FEV_1 increases achieved by albuterol up to 8 5 days *Chest* 1993;104(suppl to No. 2):112S Abstract **2.** Data on file, Boehringer Ingelheim Pharmaceuticals, Inc **3.** ATROVENT® (ipratropium bromide) Inhalation Solution full Prescribing Information

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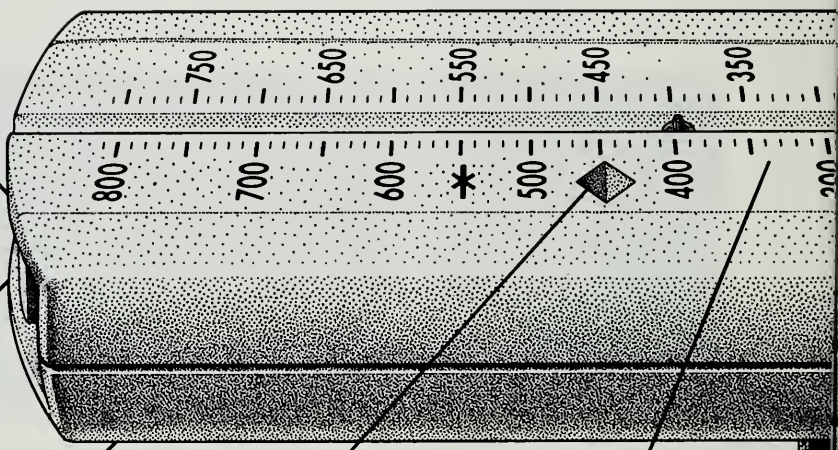

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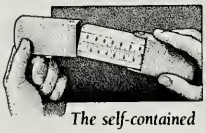

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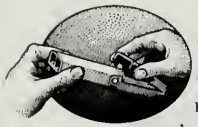

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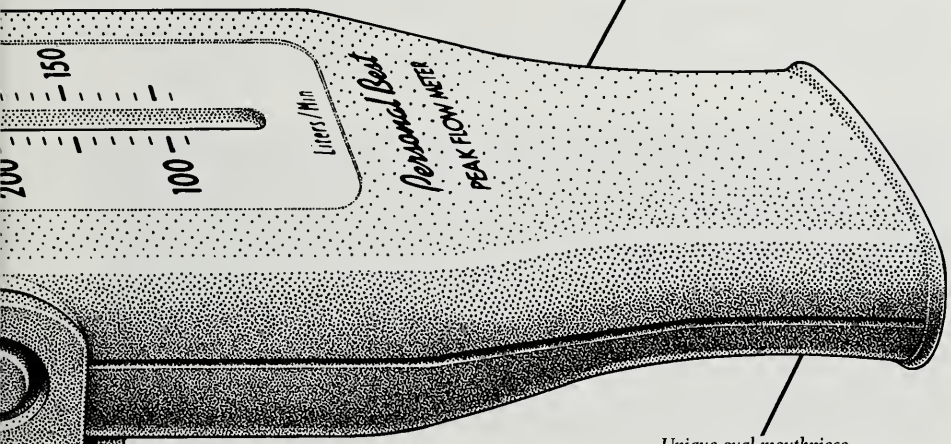

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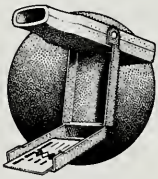

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Elevation of Systemic Oxygen Delivery in the Treatment of Critically Ill Patients—MA Hayes, AC Timmins, EHS Yau, M Palazzo, CJ Hinds, D Watson. N Engl J Med 1994;330:1717.

BACKGROUND: Elevation of systemic oxygen delivery and consumption has been associated with an improved outcome in critically ill patients. We conducted a randomized trial to determine whether boosting oxygen delivery by infusing the inotropic agent dobutamine would improve the outcome in a diverse group of such patients. METHODS: On the basis of previously published recommendations, we established the following goals: a cardiac index above 4.5 liters per minute per square meter of body-surface area, oxygen delivery above 600 ml per minute per square meter, and oxygen consumption above 170 ml per minute per square meter. If these goals were not achieved with volume expansion alone, patients were randomly assigned to a treatment or control group. The treatment group received intravenous dobutamine (5 to 200 μg per kilogram of body weight per minute) until all three goals had been achieved. Dobutamine was administered to the control group only if the cardiac index was below 2.8 liters per minute per square meter. RESULTS: A total of 109 patients were studied. In nine patients the therapeutic goals were achieved with volume expansion alone; all nine patients survived to leave the hospital. Fifty patients were randomly assigned to the treatment group, and 50 to the control group. During treatment, there were no diflerences between the two groups in mean arterial pressure or oxygen consumption, despite a significantly higher cardiac index and level of oxygen delivery in the treatment group ($p < 0.05$). Although the predicted risk of death during hospitalization was 34% for both groups, the in-hospital mortality was lower in the control group (34%) than in the treatment group (54%) ($p = 0.04$; 95% confidence interval, 0.9 to 39.1%). CONCLUSIONS: The use of dobutamine to boost the cardiac index and systemic oxygen delivery failed to improve the outcome in this heterogeneous group of critically ill patients. Contrary to what might have been expected, our results suggest that in some cases aggressive efforts to increase oxygen consumption may have been detrimental.

Tuberculosis in Young Adults and the Elderly—M Korzeniewska-Kosela, J Krysl, N Müller, W Black, E Allen, JM FitzGerald. Chest 1994;106:28.

OBJECTIVES: To compare the clinical, bacteriologic, and radiologic features of pulmonary and pleural tuberculosis in young adults and the elderly and determine if any differences exist between both groups. DESIGN: Prospective recruitment of all patients diagnosed as having pulmonary and pleural tuberculosis in British Columbia, Canada. SETTING: A population-based sample from a provincial control program TB registry. PATIENTS: A total of 218 consecutive patients whose conditions were diagnosed between January 1990 and May 1991. We excluded 15 HIV-positive patients whose conditions were diagnosed during this study. INTERVENTION: Standardized data collection of symptoms, bacteriology, and review of radiology by two readers blind to the clinical and epidemiologic data. MAIN RESULTS: There were 142 young adult patients and 76 elderly patients. The young adults had a mean age of 41.2 years and the elderly group had a mean age of 75 years of age. Fever ($p = 0.002$) and night sweats ($p = 0.02$) were more common in young adults. In culture-proven disease, hemoptysis, fever, and cough were more common in young adults ($p = 0.03$, 0.02, and 0.01, respectively). There was no difference in the duration of symptoms between the two groups. The odds ratio for cancers other than lung cancer, 3.98 (confidence interval, 1.49, 10.65) in the elderly group was the only significant risk factor to differ between the two groups. Skin test responses to 5TU PPD were positive in 86.2% of young adults and 67.6% of elderly patients tested ($p = 0.03$). A total of 79.6% of young adults and 88.15% of the elderly patients (not significant) were culture positive. Comparison of radiologic findings in young adults vs elderly patients showed no significant differences apart from those with miliary TB 0.7% vs 6.7% ($p = 0.04$). CONCLUSIONS: In this population-based study, young adults were more likely to have hemoptysis, fever, and cough and to have a positive PPD response. Cancer was significantly associated as a risk factor in the older age group. There was no difference in bacteriologically proven disease or radiologic findings between the two groups, apart from the more common occurrence of miliary TB in the elderly.

Prevalence of Hepatitis C Antibodies in Clinical Health-Care Workers—J Zuckerman, G Clewley, P Griffiths, A Cockcroft. Lancet 1994;343:1618.

Health-care workers are known to be at risk from occupational transmission of blood-borne viruses, including hepatitis C. There may be serious implications following infection with hepatitis C including possible transmission to patients. We determined the prevalence of hepatitis C virus (HCV) antibodies among health-care workers at risk of occupational contact with blood and body fluids and among source patients in reported blood-exposure incidents. Anonymised stored blood samples from health-care workers immunised against hepatitis B virus since 1991 (n = 1053) and blood samples from source patients in needlestick injuries (retrospective and prospective) since 1989 (n = 373) were analysed. 3 (0.28%) of the serum samples from health-care workers were found to be anti-HCV-positive. 17 (8.5%) of 200 source patients tested retrospectively between January 1989 and January 1992, and 24 (13.9%) of 173 source patients tested prospectively between January 1992 and June 1993 were anti-HCV-positive. During the second period, 15 (10.6%) of 142 source patients tested for human immunodeficiency virus (HIV) were positive and 7 (3 .8%) of 184 source patients tested for hepatitis B surface antigen were positive. 6 of 24 (25%) HCV-infected patients were diagnosed only after the incident; for hepatitis B, 2 (33%) of patients were diagnosed after the incident, and for HIV all patients were previously diagnosed. The seroprevalence of HCV among these health-care workers is no higher than that reported in blood donors. This suggests that there has not been significant occupational transmission of HCV to these health-care workers despite the high prevalence of HCV (often covert) among source patients in reported blood exposure in the same hospital.

A Clinical Profile of Chronic Bacterial Pneumonia: Report of 115 Cases—SH Kirtland, RH Winterbauer, DF Dreis, NE Pardee, SC Springmeyer. Chest 1994;106:15.

OBJECTIVE: To review the clinical presentation, radiology, microbiology, and response to therapy of patients with chronic bacterial pneumonia. DESIGN: A retrospective analysis. SETTING: An urban tertiary care medical center. PARTICIPANTS: One hundred fifteen patients with pulmonary and/or constitutional symptoms of at least 1 month's duration with 4,000 or more colony-forming units (CFUs) of a single bacterial species identified by quantitative culture obtained via fiberoptic bronchoscopy. MEASUREMENTS: Charts were analyzed for presence or absence of any predisposing illness, symptoms at presentation, roentgenographic abnormalities, microbiologic results, findings at fiberoptic bronchoscopy, and results of therapeutic intervention. RESULTS: Sixty-five percent of patients with chronic bacterial pneumonia had a predisposing disease, 35% were "normal." Cough, fatigue, dyspnea, and weight loss were predominant symptoms in both groups. Bronchogenic carcinoma was newly diagnosed in 16 patients (14%). *Haemophilus influenzae* or alpha-hemolytic streptococcus was isolated in 68% of patients. Risk of recurrence of infection was inversely associated with duration of therapy in both groups. CONCLUSIONS: Chronic bacterial pneumonia is more common than previously recognized. It occurs in patients with and without a predisposing illness. Clinical presentation, roentgenographic appearance, and bacteriology are similar between the two groups. Cure requires prolonged antibiotic therapy.

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A class action lawsuit, *Ahearn v. Fibreboard* and a Global Settlement Agreement, have been filed in U.S. District Court in Tyler, Texas. If approved, this class action and settlement will resolve all future asbestos-related personal injury claims against Fibreboard and its predecessors, including PABCO, as well as third-party claims arising out of such claims.

A class action, *Continental Casualty Co. v. Rudd*, was also filed against two defendant classes seeking approval of a settlement entered into between Fibreboard and certain of its insurers to settle all liabilities and obligations of the insurers under insurance policies they issued to Fibreboard.

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A Laboratory Comparison of Three Pulmonary Artery Oximetry Catheters—PE Scuderi, DA MacGregor, DL Bowton, RL James. Anesthesiology 1994;81(1):245.

BACKGROUND: Measurement of mixed venous hemoglobin oxygen saturation via catheters employing reflectance spectrophotometry has been available for more than 10 yr. Despite numerous clinical reports that have presented data showing the poor accuracy of these devices when used clinically, they are still widely used in clinical care. The reason for lack of agreement with measurements made using bench spectrophotometry is unclear. The purpose of this study is to define the performance limitations of three hemoglobin oxygen saturation catheters (Oximetrix 3, SAT-2, and HEMOPRO$_2$) in a controlled laboratory environment using a blood flow loop primed with fresh whole human blood as a model. Our hypothesis is that the performance limitations of these devices represent inherent limitations in the technology, not error introduced by patient anatomy and physiology. METHODS: Blood was equilibrated in a flow loop to four analytic gas mixtures designed to achieve oxygen saturation of approximately 50%, 60%, 70%, and 80%, respectively, with carbon dioxide tension, pH, and temperature held constant. Saturation readings from the catheters were collected on-line by microcomputer. Periodic blood samples were withdrawn from the flow loop for analysis on a bench spectrophotometer and subsequent comparison with catheter-derived values. RESULTS: By all measures, performances of the Oximetrix 3 and SAT-2 systems were comparable (all data are presented as percent saturation unless otherwise noted); bias ± precision was 3.20 ± 2.47 and −1.25 ± 3.36, respectively, versus −9.97 ± 7.05 for the HEMOPRO$_2$. The 95% confidence limits based on intracatheter variability were ±3.49, ±2.90, and ±9.13 for the Oximetrix 3, SAT-2, and HEMOPRO$_2$, respectively. The 95% confidence limits based on total variability, although similar for Oximetrix 3 (±4.83) and SAT-2 (±6.59), were larger for the HEMOPRO$_2$ (±13.82). The 95% confidence intervals for agreement between catheter brands were −2.14, 11.04 (Oximetrix 3–SAT-2); −0.18, 26.52 (Oximetrix 3–HEMOPRO$_2$); and −5.24, 22.68 (SAT-2–HEMOPRO$_2$). CONCLUSIONS: While the Oximetrix 3 and SAT-2 may be acceptable as continuous monitors used to detect changes or trends, none of the three systems is equivalent to conventional bench oximetry for the measurement of hemoglobin oxygen saturation.

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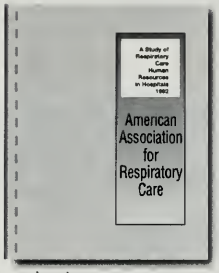

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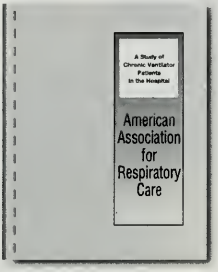

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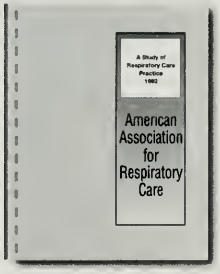

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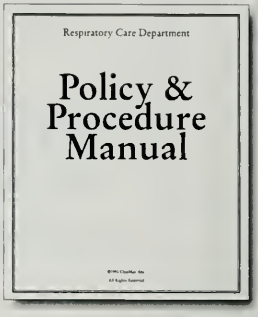

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Routine Measurement of Work of Breathing: Is It Necessary?

One of the most challenging aspects of respiratory care is weaning the long-term ventilator-dependent patient. Regardless of the cause of acute ventilatory failure, such patients consume enormous resources and increase the overall cost of patient care. Numerous predictive indices[1-5] as well as the development of various ventilatory modes[6-8] have been directed at this problem. Some authors recommend monitoring work of breathing (WOB) during management of these patients.[9,10] However, the measurements and calculations associated with WOB determination have made routine clinical use impractical and have been normally performed only during research protocols.

In this issue of RESPIRATORY CARE, Blanch and Banner[11] report an expertly performed study validating the WOB algorithms incorporated into the Bicore CP-100 bedside monitor. This monitor makes measurement of WOB much easier than with previous methodology. This device also noninvasively provides airway pressure, volume, and flow curves (as do other systems). The question still to be answered, however, is when and on whom should WOB measurements be made?

Before addressing this question, a few comments about esophageal balloons are in order. The placement of an esophageal balloon in a critically ill patient weaning from ventilatory support is not without problems. We have found it extremely difficult if not impossible to place standard esophageal balloons in some patients when an artificial airway is present and the patient does not or cannot voluntarily cooperate (ie, swallow). The posterior displacement of the esophagus makes entry difficult, whereas entry into the trachea itself is facilitated. On occasion, we have noted near perfect matching of esophageal and airway pressure following balloon insertion, indicating tracheal placement. Once the balloon is placed, proper position must be assured either by having the patient inspire against an occluded airway[12] or by determining the presence of cardiac contractions on the esophageal pressure tracing.[13] The incorporation of an esophageal balloon on nasogastric tubes does make placement considerably easier.

Blanch and Banner[11] argue for the measurement of total WOB, which requires the measurement of chest-wall compliance. With an esophageal balloon in place, the measurement of chest-wall compliance is easy in the relaxed mechanically ventilated patient. However, in the spontaneously breathing patient it is impossible. In this setting, Blanch and Banner[11] recommend the use of 1 to 2 mg midazolam to achieve the muscle relaxation necessary for measurement followed by reversal with flumazenil (about 0.2 mg). This procedure is costly and potentially dangerous (rendering the patient unable to breathe spontaneously), and we would argue that the data add little if anything to the measurement of lung work. In a given patient, chest-wall compliance is usually a constant, changing little (if any) over time. Exceptions to this are patients with muscular hypertonicity or severe chest-wall burns or who are undergoing peritoneal dialysis. However, weaning is usually not considered until such acute conditions have been resolved. As a result, assuming a constant value for chest-wall compliance adds a systematic error that should not change over the course of ventilatory support. If the routine use of actual chest-wall compliance is necessary for the calculation of total WOB, we believe its measurement should be performed each time WOB is calculated—not simply once during the course of ventilatory support!

Is the routine measurement of WOB ever necessary? First of all, WOB is a poor estimate of patient effort. WOB is determined by the pressure required to move a volume of gas into the lungs. If no volume is delivered, then no work is performed despite a maximal effort. For example, no work is performed during a maximal inspiratory pressure (MIP) maneuver, even though patient effort is maximal. If you push against a wall all day but do not move it, you will be exhausted at the end of the day, but will not have performed any work! Because of this problem, we have found monitoring the change in esophageal pressure (ΔP_{es}) more useful than WOB determination during the titration of ventilatory support. Also, change in esophageal pressure with adjustment of the ventilator is easier for practitioners to comprehend than a change in joules/liter or joules/min. We have found monitoring ΔP_{es} relative to MIP useful in some patients because a ΔP_{es} greater than about 40% of MIP produces fatigue in many persons.[3] Pressure support levels can be targeted to a ΔP_{es} that is unlikely to produce fatigue. Because baseline effort in chronically ill patients may vary greatly from textbook normals, we believe that monitoring change in esophageal pressure over time or with a change in mode of ventilatory support is far more useful than the actual numerical value of total WOB.

For routine clinical assessment of altered levels of ventilatory unloading during weaning, the the change in central venous pressure (ΔCVP) is a commonly available alternative to esophageal pressure as an indicator of intrapleural pressure changes.[14-16] Respiratory variation in the CVP reflects pleural pressure changes as well as do esophageal pressure changes. Bedside display of CVP and observation of variation during the respiratory cycle allows easy assessment of the effects of ventilator settings on patient breathing effort. In addition, clinical signs of increased patient effort (accessory muscle use, retractions, rapid shallow breathing) can be nicely related to changes in respiratory CVP variations.

We have previously proposed the answering of three questions regarding the evaluation of monitoring technology:[17] Is it technically possible? Is it clinically useful? Is it cost-effective? Although monitoring WOB with the Bicore CP-100 is technically possible, its routine clinical usefulness has not been established. Will it decrease the number of days of mechanical ventilation? If it decreases the number of ventilator days, then it may be cost-effective. But, until this has been demonstrated, routine use of total WOB measurements must be questioned.

Work of breathing measurements are essential for research and are useful for teaching but in our opinion unnecessary for routine weaning of ventilator-dependent patients. We do place esophageal balloons in long-term ventilator-dependent patients weaning from ventilatory support—but only when progress is inconsistent with clinical presentation.

Embracing new technology should only occur when improvement in patient management can be demonstrated. In today's cost-conscious environment, we must focus on the effect of new technology on outcome and not be seduced by our love of technology for the sake of technology.

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Work of Breathing: How To Define and When To Measure?

Critical to providing optimal care for the mechanically ventilated patient is an understanding of the concept of work of breathing (WOB). But which component of total WOB is important: mechanical, elastic, resistive, chest wall, lung, thoracic, or imposed work of the ventilator? The point is *work of breathing can be an extremely complex topic.* Distinguishing between these components requires an understanding of the ventilator pressures, patient pressures, and definitions for work. It is also important to be aware of efficiency and the respiratory muscle load as partially reflected by the effort to breathe. For each work component there is a defined, measurable pressure-volume area, but which pressure differences are being measured for which work? Whereas work defines a pressure-volume area (generated by the ventilator and/or patient), patient pressure-time product or effort can also reflect a measure of efficiency (work accomplished compared to energy used). Work measurements and their components have been clearly and expertly defined elsewhere,[1-5] yet the term work continues to be used loosely. Commonly, an effort is called work (as in the trigger effort) or work terms are bundled or mislabeled. The problem becomes somewhat clearer when one considers the various work measurements and the monitoring required—airway pressure, plateau pressure, maximum airway pressure, auto-PEEP, esophageal pressure, chest-wall compliance, and lung compliance. Because there are two distinct sources for generating gas flow, it is helpful to separate the ventilator and patient-work issues.

First, the ventilator serves as a controllable, comparatively limitless source of power for lung inflation. Its pressure generation can indicate the work load of breathing,[6] and a difference between the airway pressure-volume area of a passive, controlled breath and an assisted breath can be considered an index of work.[7,8] Of special concern in ventilator work/effort studies have been the role of the endotracheal tube,[9-11] the effects of circuitry,[12] and the role of the demand valve.[13,14] Studies have been conducted on ventilator synchrony with specific modes[15,16] and with differences among flow systems.[17] While differences among ventilators exist, manufacturers appear to have responded to many concerns, and controllable differences among modern ventilators may be insignificant and may not affect outcome.[18] On the other hand,we can anticipate that matching ventilator delivery (as in pressure support ventilation and proportional assist ventilation) to patient drive and patient waveforms will improve synchrony, more smoothly allow patient assumption of work, and reduce unnecessary pressure exposure. Therefore, it is now justified and prudent to examine airway pressure tracings carefully, with a particular concern for the role of ventilator mode and the effect of ventilator circuitry on the work or effort expended by the patient.

Second, and perhaps of more importance in the difficult-to-wean patient, are not only the ventilator pressures, mode, and equipment but also awareness of the depth, duration, and endurance of patient-generated pressures.The patient's ability to generate pressures to sustain his own breathing is often accurately assessed (but not actually measured) by the degree of disease resolution and the inference of work and effort by measurement of breathing frequency (f), tidal volume (V_T), f/V_T, negative inspiratory force, vital capacity, maximum ventilatory volume, and blood gas values. In the difficult-to-wean patient, more sophisticated assessments may be made—such as occlusion pressure[19] or work estimates from airway pressure-volume scalloping.[7,8] Measurements thought to broadly and more precisely determine weanability (work, efficiency, and the respiratory work load) have been the cost of breathing (V_{O_2COB}) and esophageal pressure-time product (PTP_{es}). Shikora et al[20] reported that a V_{O_2COB} of less than 15% was found to accurately predict weaning success in 20 patients. As a global measure, V_{O_2COB} would seem to be an excellent weanability index but, unfortunately, V_{O_2COB} is difficult to measure, has not been extensively studied, and may not be reliable.[21,22] PTP_{es} should measure work and a degree of its efficiency, but its measurement during wide esophageal pressure swings is problematic because determining the start and stop points for measurement has not been standardized. In 1958, Campbell introduced the definition of mechanical work as an esophageal-pressure volume area between the zero flow points of inspiration and including the pressure necessary to expand the chest wall.[1] This measurement has elastic and resistive components and may be of value in determining the patient's potential role in reloading the respiratory muscles. Therefore, compared to V_{O_2COB} and PTP_{es}, mechanical work is more clearly defined and more easily measured but it, also, has not been studied extensively.

In this issue Blanch and Banner[23] report an excellent

correlation and agreement between a commercial device to measure work and a dedicated work measurement system used in a research setting. This bench study has clinical implications. Its clinical use requires, of course, a simple-to-place esophageal balloon-catheter that causes less discomfort than a nasogastric tube and is probably accurate even when placed next to a nasogastric tube.[24]

This work measurement, however, has limitations of accuracy and interpretation. The measurement itself has a low signal-to-noise ratio due to position, swallowing and movement artifacts, and cardiac oscillations. Related to its position and its value representing the span of pleural pressures, it does not directly reflect the true pleural pressure, the true driving pressure, but it is the best estimate available. Also, as previously stated, the measurement does not, by definition, measure esophageal pressure changes that do not involve movement of a volume of gas—such as occurs in an effort to trigger the ventilator. Finally, the work measured from a ventilator-assisted breath would not reflect the energy used by the patient.[25] In spite of these potential drawbacks, a critical observer should be able to discern consistent work measurements.

Because the concept of work is so important to ventilator management, it is incumbent upon care pro-viders to discuss (and insist that others discuss) work precisely as a measurable parameter. There is much yet to be studied in the area of ventilator issues, patient-related work issues, and the coordination of sharing work towards the goal of weaning. Without outcome studies showing the actual rather than suspected benefit of work measurements, routine determination of work cannot, as yet, be recommended. In an approved protocol, many questions about work require further investigation. With physicians, RCPs, and nursing personnel who understand the value and limitations, a work measurement can assist in managing the difficult-to-wean patient. Therefore, how should work be defined and when should it be measured? Measuring work in the difficult-to-wean patient by the Campbell-definition may provide insight into weanability, and equipment has become available for such measurements.

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A Comparison of Three Methods of Metered Dose Bronchodilator Delivery to a Mechanically Ventilated Adult Lung Model

Robert Harwood MSA RRT, Joseph L Rau PhD RRT, and Lynda Thomas-Goodfellow MBA RRT

INTRODUCTION: Metered dose inhaler (MDI) reservoirs are commonly used in the hospital to deliver MDI medications to intubated patients on mechanical ventilation. The purpose of this study was to evaluate 3 methods of MDI delivery through an adult mechanical ventilator circuit. MATERIALS & METHODS: A Siemens Servo 900C in the volume-control mode was used to ventilate a lung model with standardized settings: $\dot{V}_E$ 9.6 L, respiratory rate 12 breaths/min, t_I 20% (1 s), and an accelerating flow waveform (a modified ramp flow waveform). An 8-mm-ID endotracheal tube (ETT) was attached to a rubber test lung (TL). The humidifier was bypassed for all trials. Aerosolized albuterol was delivered by MDI using 3 different methods. Method 1—MDI was attached directly to the ETT between the Y and 15-mm adapter using the Medicomp actuator/adapter. Method 2—MDI was attached to the Monaghan Aerovent reservoir and placed on the inspiratory limb just proximal to the patient Y. Method 3—MDI was attached to the Aerosol Cloud Enhancer (ACE), positioned as in Method 2. Aerosol drug was captured at the distal ETT tip and was measured spectrophotometrically. The amount of drug at the end of the ETT was expressed as a percent of the measured output of the MDI. Data were analyzed using repeated-measures analysis of variance and Scheffe's test. RESULTS: Method 1 delivered 1.69%, Method 2 delivered 21.4%, and Method 3 delivered 46.3% of the total dose to the end of the ETT. The 3 methods differed significantly ($p < 0.001$). Pairwise post-hoc comparisons showed that each method differed significantly from the other two methods ($p < 0.05$). CONCLUSIONS: In an intubated adult-lung model being mechanically ventilated, the use of a reservoir device on the inspiratory limb of the ventilator circuit increases the percent of dose reaching the end of ETT. The DHD ACE delivered a significantly greater percent of dose as compared to the other two methods examined. [Respir Care 1994;39(9):886-891.]

Mr Harwood is Assistant Professor, Dr Rau is Professor and Chair, and Ms Thomas-Goodfellow is Director of Clinical Education, Department of Cardiopulmonary Care Sciences, Georgia State University, Atlanta, Georgia.

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Introduction

Aerosolized delivery of drugs offers a convenient route of administration for agents targeted to the respiratory tract and is a mainstay for patients with respiratory disease. Mechanically ventilated intubated patients are frequently candidates for such aerosol therapy because of pre-existing or acute pulmonary disease. Two common methods of administering drugs by aerosol to patients on mechanical ventilators are small volume nebulizers (SVN) and metered-dose inhalers (MDI). Previous studies have evaluated the use of nebulizer systems for aerosolization using both in-vitro and in-vivo designs, and have measured both clinical effects and quantitative deposition.[1-8]

An MDI offers an alternative to nebulized aerosol delivery. Compared to the enteral route, inhalation of beta-adren-

ergic agonists from an MDI provides more rapid onset of action, greater bronchodilation, more desirable therapeutic/toxic ratio, and fewer side effects.[9,10] Additionally, cost-savings of therapist time and patient charges may be realized with use of MDIs for aerosol treatments with intubated patients as well as non-intubated ones.[1,11-13] External gas sources required by an SVN are not needed for an MDI. External gas flows introduced into the patient ventilator circuit adversely affect the function and monitoring systems of the ventilator. Continuous flow from an SVN can cause difficulty in triggering breaths during pressure support for some patients.[14] In infants, the addition of a continuous-flow-driven SVN can result in marked increases in peak inspiratory pressure and tidal volume.[15] Opening the circuit to place the SVN in-line has been shown to be a source of airway contamination.[16]

Despite the advantages of aerosol administration using an MDI, evidence suggests that the endotracheal tube (ETT) and its adapter act as an efficient baffle reducing the amount of drug reaching the lung.[1,4,12,17-19] In addition, the size of the ETT, the exact method of administration, and the type of MDI actuator/adapter used can all influence aerosol delivery and deposition in the lung.[17,20,21]

In our experience, clinicians often administer MDI bronchodilator agents to intubated patients with either an actuator/adapter or spacer device. We have shown that use of a commercially available device (Monaghan Aerovent reservoir*) significantly increases aerosol drug delivery with a ventilated intubated model.[22] Recently, a new spacer called the Aerosol Cloud Enhancer (ACE) was introduced. This study was conducted to compare the relative efficiency of MDI aerosol delivery among an ETT actuator/adapter, the ACE reservoir, and the Aerovent reservoir in a mechanically ventilated adult lung model.

Materials and Methods

Study Design

An in-vitro model was used in the investigation. A Siemens Servo 900C in volume-control mode was employed to ventilate a lung model with standardized settings: $\dot{V}_E$ 9.6 L, respiratory rate 12 breaths/min, t_I 20% (1 s), and the accelerating-flow waveform. There was no pause time or PEEP. A disposable adult ventilator circuit was attached to an 8.0-mm-ID ETT. The distal end of the ETT was cut off above the Murphy eye in order to make better contact with the cotton filter placed there to capture the aerosol. The cotton was placed in an adapter connecting the ETT to a standard test lung (TL). The end of the ETT was inserted into the adapter with the distal tip resting directly against the cotton. The ETT-TL was allowed to hang unsupported from the ventilator circuit. The ventilator's humidifier was bypassed for all trials. Aerosolized albuterol was delivered by MDI using three different methods. Method 1—MDI was adapted directly to the ETT, distal to the patient Y, using the Medicomp MDI actuator/adapter (Fig. 1A). Method 2—MDI was used with the Aerovent reservoir placed on the inspiratory limb just proximal to the patient Y (Fig. 1B). Method 3—ACE was positioned as in Method 2 (Fig. 1C).

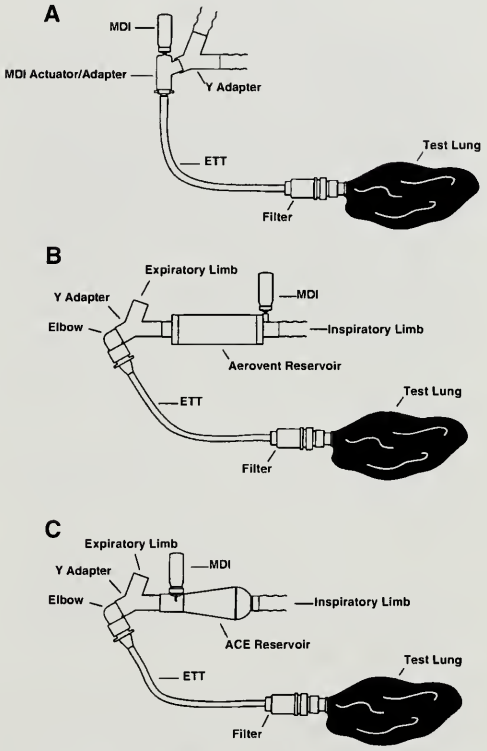

Fig. 1. Illustration of the 3 methods used to administer aerosolized bronchodilator to an intubated adult lung model. (A) The MDI is situated near the ETT with no spacer. (B) The MDI is positioned in the inspiratory limb distal to a spacer/reservoir device—Aerovent. (C) The MDI is positioned as in B but the reservoir device in this model is the aerosol cloud enhancer—ACE.

Seven MDI canisters were used to deliver aerosolized drug by each method. To minimize variability due to the canisters, the same seven canisters were used for all three methods of administration. All canisters were full. In each method, the canister was shaken to mix the contents and discharged once to prime the valve prior to the measure-

*Manufacturers and suppliers are identified in the Product Sources section at the end of the text.

ment run. The canister was also shaken prior to each discharge at the beginning of inspiration. Each discharge was timed to occur 1 second prior to the beginning of inspiratory flow from the ventilator, to maximize delivery as suggested by Crogan and Bishop's study.[17] Following each activation, we allowed a pause of 6 breaths or 30 seconds, until a total of 10 discharges had been delivered. All MDI activations were performed by the same operator to reduce the variability due to differences in operator technique.

Measurement

The quantity of drug delivered was determined using a spectrophotometer. This technique has been described in a previously published study.[22] A standard solution of the drug (0.049 mg/mL) was prepared by dissolving pure albuterol base in 90% ethanol. This solution had an absorbance (A) of 0.42 nm at 279 nm, a characteristic absorbance peak from which unknown concentrations of the drug were calculated. Following the tenth discharge, the cotton was extracted from the distal end of the ETT and placed in a test tube containing 9 mL of 90% ethanol. The cotton was agitated with a stirring rod to dissolve the drug powder. The cotton wadding was then removed from the test tube and squeezed to remove the excess solution. The solution was then centrifuged to "spin down" cotton fibers found in the solution. A portion of the centrifuged solution was then placed in a cuvette and measured.

Absorbance of the filtered solution was measured at 279 nm, and the amount of drug was calculated from the standard solution's concentration and absorbance as follows:

$$C_{samp} = \left(\frac{A_{samp}}{0.42\ nm} \right) \times 0.049\ mg/mL,$$

where C_{samp} is the concentration of the sample in mg/mL and A_{samp} is the absorbance of the sample in nm. The values 0.42 nm and 0.049 mg/mL are the absorbance and concentration of a standard solution of the drug.

A sample series of solution washed from the cotton alone (with no drug) using 9 mL of solvent was also measured and its absorbance was 0.05 nm. This value was subtracted from the measured absorbance of the drug solution for all trials of all three methods. The total dose for each MDI was measured by discharging the canister directly into cotton placed inside the Medicomp actuator/adapter, which was in-line between the ventilator circuit Y-connector and the ETT. This total dose was measured in-line with positive pressure breaths to control for any possible effect of pressure on the amount of drug released from the canister or on particle size. The percent of drug delivered at the end of the ETT was calculated as the amount recovered at the bottom of the ETT divided by the measured MDI dose. In a previous study,[22] we determined the efficiency of the cotton in capturing the drug by placing a second cotton filter immediately after the first at both the proximal and the distal points of the ETT. The second cotton filter showed no trace of the drug after correction for the absorbance of the extract of cotton alone.

Data Analysis

Differences among the delivered drug amounts among the 3 methods of administration were analyzed using a repeated-measures analysis of variance with pairwise follow-up comparisons using Scheffe's test. A significance level of 0.05 was chosen.

Results

The percent of drug delivered to the end of the ETT from each MDI canister with each method is shown in Table 1. Method 1 delivered 1.69% (range 0.65-3.48%), Method 2 delivered 21.40% (range 16.1-26.6%), and Method 3 delivered 46.30% (range 37.1-55.7%). Delivered drug was significantly different among the three methods ($p < 0.001$), and each method was significantly different from the other two ($p < 0.05$).

Discussion

In non-intubated patients using an MDI alone, deposition of aerosol is affected by the anatomy of the airway, ventilatory pattern, flowrate, and particle size. One study has shown that 10 to 15% of the dose is deposited in the lung and 80% in the oropharynx.[23] With the addition of a spacer, the amount deposited in the lungs increases to 21% and in the oropharynx to 16%, with 56% remaining in the spacer.[23]

Intubated patients fare no better when MDI aerosol is delivered without a spacer. Our 1992 study[22] showed that the total dose of a bronchodilator aerosol delivered by MDI without a spacer to the end of an ETT was 7.3%. On inspection, a large quantity of drug powder was seen on the actuator/adapter and ETT adapter. A high percentage of the loss of aerosolized drug in intubated patients using an MDI attached directly to the ETT occurs at the ETT adapter and in ETT itself.[22] This is not surprising because the aerosol actuation expels the droplets at a high initial velocity. This, along with large-sized droplets, causes impaction and deposition on the narrow ETT adapter. Turbulent airflow resulting from curvature of the ETT and the relatively small internal diameter of the ETT also contribute to impaction and premature deposition of aerosol.[6,17] As particles enter a reservoir, their initial velocity is reduced, and their size decreases, facilitating deposition in the lungs.[24] Our 1992 study[22] showed further

that with the addition of a reservoir to the inspiratory limb placed proximal or distal to the Y adapter, deposition increased to 32.1% and to 29%, respectively.[22] On inspection, only a small amount of white powder was seen on the ETT adapter and in the ETT itself.

Although the presence of an ETT causes considerable loss of aerosol from an MDI, it also provides an advantage. The ETT eliminates the absorption of drug in the oropharynx, thereby reducing systemic effects.[6,17] This is especially important in patients using steroids because larger doses may be delivered to the parenchyma with less risk of systemic effects. Because the percent of total dose delivered is shown to increase with use of a reservoir device placed on the inspiratory limb of the ventilator circuit, we suggest that MDI dose (number of puffs) be titrated to patient response while monitoring for side effects.

The shape of the spacer is also important in delivery of drug to the end of the ETT. Lindgren and colleagues[25] compared tube-shaped and pear-shaped spacers and found a pear-shaped spacer to be significantly better than the tube-shaped one. Newman and colleagues[26] demonstrated that greater deposition of radiolabeled aerosol was achieved with a pear-shaped spacer than with a tube-shaped spacer. A study by Moren[27] concluded that pear-shaped spacers appeared to be most effective in increasing drug delivery to the airways of non-intubated subjects. We are not aware of any studies comparing tube-shaped spacers to pear-shaped spacers in intubated, mechanically ventilated adult patients.

An unpublished study (personal communication, D Williams RRT, University of Kansas Medical Center, June, 1992) looked at particle size distribution and delivery of aerosol from various reservoir devices using Atrovent. In Williams' study, the ACE delivered a greater amount of medication in the respirable particle size range than the Aerovent reservoir. Ebert and colleagues[28] found that reservoir chambers had a greater mean available respirable volume (MARV) than in-line MDI adapters and elbows. In Ebert's study of three types of reservoir chambers, the ACE had the greatest MARV of the three chambers tested using three commercially available MDI steroid preparations.

Our bench study used a simulated lung model with ventilator settings that would be appropriate for an adult patient receiving mechanical ventilation. We did not manipulate controls during the study, such as adding PEEP, changing percent of pause time, adding pressure support ventilation, or changing the mode of ventilation. Hess and co-workers[29] evaluated the effectiveness of an MDI with mechanical ventilation and found that aerosol deposition at the end of the ETT was not significantly affected by changes in tidal volume and flowrate. Flavin and colleagues[20] found ventilator settings had little to do with aerosol delivery. The humidifier was bypassed according to the results of O'Riordan and colleagues.[30]

We recognize that our results from this study differ from those of our previous study.[22] In that study, the MDI placed directly on the ETT delivered 7.3% of the total dose, and the Aerovent chamber placed on the inspiratory limb delivered 32.1%. The smaller amount of delivered drug in the present study may be due to differences in filtration techniques, differences in sample lots of the devices used, or both. In our 1992 study,[22] we filtered the solvent-drug solution to remove cotton fibers, which may have allowed some evaporation of the alcohol solvent and resulted in a slightly more concentrated residual solution. Use of the centrifuge instead of filtration allowed the container of drug solution to remain closed, possibly reducing the loss of solvent.

The results from a study by Fuller and colleagues[31] also differ from ours. In their study of mechanically ventilated patients, Fuller showed a range of deposition of 5.53-6.33% using chamber spacers. These values are lower than ours and may indicate the degree to which clinical variables can affect aerosol delivery to ventilator-supported patients.

One facet of this study that challenged us was coordinating the MDI actuation with the delivered flow from the ventilator. Although the same person actuated all MDIs in the study, a few trial runs were needed prior to testing to coordinate the timing between actuation and ventilator flow. Because the ventilator was set in volume control and at a set rate, standardizing the coordination was relatively easy. However, in the clinical setting, timing the actuation could be difficult. Timing will vary among caregivers who administer MDIs and thereby alter the dose delivered. Patient triggering of the ventilator and spontaneous breathing through the ventilator circuit make timing the actuation of an MDI an even greater challenge.

An obvious difference between the ACE reservoir and the Aerovent reservoir is the Aerovent's ability to collapse within the patient circuit. Reusing the Aerovent reservoir is a simple matter of unfastening and re-expanding it. In our experience, this offers a convenience to practitioners in the ICU. It also reduces the possibility of contamination of the ventilator circuit by limiting the number of times the ventilator circuit is opened to the room—an important consideration as the intervals between ventilator circuit changes are lengthened. A potential problem exists with the Aerovent reservoir. The chamber is flexible and could cause the jet nozzle to become misaligned with the exit port, increasing aerosol loss due to impaction on the chamber. In our study, the chamber's position was maintained so that misalignment could not occur.

The ACE reservoir is somewhat heavier than the Aerovent and, when left in the circuit, may pull slightly on the inspiratory limb of the circuit and the ETT causing ventilator disconnection and/or inadvertent extubation.

Therefore, it may be prudent to remove the reservoir after each treatment unless proper support of the Y-adapter and circuit can be ensured. We did not study the effects that the ACE might have on ventilator function by leaving it in the circuit. However, such studies are warranted.

When we questioned therapists on their choice of reservoirs for ventilator circuits, ease of use was cited as a major factor in deciding which reservoir device to use.

Conclusions

The use of a reservoir device increased the percent of dose reaching the end of an ETT when albuterol was delivered to a mechanically ventilated adult lung model via MDI. We showed that use of the ACE reservoir resulted in a larger dose delivered to the end of an ETT than was delivered using either an ETT adapter/actuator or an Aerovent reservoir. In-vivo response studies are needed to further investigate the effect of reservoir devices on aerosol delivery to patients receiving mechanical ventilation.

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PRODUCT SOURCES

MDI Reservoirs:
Aerosol Cloud Enhancer (ACE), 11-1010 DHD-Diemolding Healthcare Division, Canastota NY
Aerovent Aerosol Holding Chamber, Monaghan Medical Corp, Plattsburgh NY

MDI Adapter:
Medicomp Actuator/Adapter, Medicomp Co, Coon Rapids MN

Bronchodilator:
Albuterol sulfate (Proventil), Schering Corp, Kenilworth NJ

Spectrophotometer:
Beckman Model DU 640 Spectrophotometer, Beckman Instruments Inc, Fullerton CA

Endotracheal Tubes:
Armstrong Medical Products, Lincolnshire IL

Circuit Filters:
Pall Breathing Circuit Filter BB-50T, Glen Cove NY

Ventilator:
Siemens Servo 900C, Siemens Life Support Systems, Piscataway NJ

Centrifuge:
IEC Centra-4B Centrifuge, Needham Heights MA

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Flow-Triggering Does Not Decrease the Work of Breathing and Pressure-Time Product in COPD Patients

Kelly Jager RRT, Martin Tweeddale MB PhD, and Tammy Holland RRT

BACKGROUND: Because of their resistance, CPAP systems, triggered by a preset pressure change (PT-CPAP), impose a greater work of breathing (WOB) than is imposed by endotracheal tubes and circuitry alone. Flow-triggered CPAP (FT-CPAP) has been developed to avoid some of this increase. METHOD: With a pulmonary mechanics monitor, we measured and compared WOB and pressure-time product (PTP) with FT-CPAP and PT-CPAP (in the Puritan-Bennett 7200ae) in 7 hemodynamically stable COPD patients who were ready for weaning. Significance of differences in results between the two systems was tested by 2-way repeated-measures analysis of variance. RESULTS: Mean (SD) WOB on FT-CPAP was 1.19 (0.31) J/L and on PT-CPAP 1.24 (0.29) J/L ($p = 0.79$). Mean PTP on FT-CPAP was 251 (88) cm $H_2O \cdot s \cdot min^{-1}$ and on PT-CPAP 282 (96) cm $H_2O \cdot s \cdot min^{-1}$ ($p = 0.81$). No difference in sensitivity as determined by the monitor was seen between PT-CPAP and FT-CPAP. CONCLUSION: Our results do not support the superiority of FT- over PT-CPAP in COPD patients being weaned from mechanical ventilation. [Respir Care 1994;39(9):892-896.]

Background

It has been shown that ventilator continuous positive airway pressure (CPAP) systems that are triggered by a preset pressure change (pressure-triggered or PT-CPAP) increase the patient's work of breathing (WOB) in addition to that imposed by endotracheal tubes and breathing circuitry.[1-3] To compensate for some of this increased work, flow-triggering has been made available on the Puritan-Bennett 7200ae ventilator.* With this option operative (called Flow-By by the manufacturer), the practitioner sets a continuous base flow of 5-20 L/min and a flow sensitivity of 1-10 L/min. Flow sensitivity cannot exceed 50% of the baseline flow. When the patient's inspiratory effort causes the continuous flow traversing the expiratory flow sensor to fall by the amount of the flow sensitivity setting, the demand valve opens, providing up to 180 L/min of flow for patient inspiration. During inspiration, the ventilator attempts to maintain pressure in the system at 1.5 cm H_2O above the CPAP setting. When flow traversing the expiratory flow sensor exceeds flow through the inspiratory flow sensor by more than 2 L/min, exhalation begins. At the start of exhalation, baseline flow is reduced to 5 L/min until expiratory flow ceases. At that point, baseline flow returns to its set value.

Some studies have shown a decrease in WOB with FT-CPAP compared to PT-CPAP,[4-7] whereas one study has shown no difference.[1] However, the experimental methods, study populations, and measured variables have dif-

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*Manufacturers and suppliers are identified in the Product Sources section at the end of the text.

fered among studies, making it difficult for the clinician to determine the utility of FT-CPAP. Only one published study has investigated FT-CPAP in intubated ICU patients with COPD.[4] Because of these differing results, we thought it important to determine whether FT-CPAP provided by the Puritan-Bennett 7200ae has potential benefits over PT-CPAP in ICU patients who are being weaned from mechanical ventilation. We therefore compared WOB and pressure-time product (PTP) between FT-CPAP and PT-CPAP in a group of ICU patients with a history of COPD.

Methods

Patients were enrolled in the study if they met the following inclusion criteria: (1) had a history of COPD with CO_2 retention; (2) were hemodynamically stable without inotrope support; (3) were in the recovery phase from an acute event; (4) were adequately ventilated (to a normal pH) on pressure support < 10 cm H_2O; (5) were being monitored with a pulmonary mechanics monitor with an esophageal balloon in place, providing data to facilitate ventilator weaning; and (6) gave evidence of spontaneous respiratory function adequate to permit unassisted breathing for the duration of the protocol. For interface with the pulmonary mechanics monitor, a pneumotachograph was inserted between the endotracheal tube adapter and the breathing circuit, and an esophageal-balloon catheter was passed to the distal esophagus via the nose. Correct placement of this catheter was verified by the Mueller manoevre[8] and a chest radiograph.

Prior to the study, patients were placed on the Puritan-Bennett 7200ae. Patients were studied in the semi-Fowler position with a pulse oximeter in place; bronchodilators were withheld for the duration of the study, and no suctioning took place during the study. The base flow was set at 20 L/min with sensitivity at 3.0 L/min. Positive end-expiratory pressure (PEEP) was unchanged throughout the study.

At the beginning of the study, patients were rested for 20 minutes by using the assist-control mode to achieve a WOB < 0.3 J/L. (We increased pressure support ventilation to achieve the same low WOB in one patient who could not tolerate the assist-control mode.) Patients were then randomly assigned to a 15-minute period of either FT- or PT-CPAP. After a second 20-minute rest period with WOB < 0.3 J/L, each patient was changed to the other CPAP for another period of 15 minutes; a final 20-minute rest period completed the study phase (Table 1).

Table 1. Sequence and Duration of Events within the Study Protocol

Group 1	Group 2	Duration (min)
Rest period	Rest period	20
PT-CPAP	FT-CPAP	15
Rest period	Rest period	20
FT-CPAP	PT-CPAP	15
Rest period	Rest period	20

Breath-by-breath tidal volume, respiratory rate, sensitivity, intrinsic PEEP ($PEEP_i$), WOB, and PTP were obtained from the monitor. The sensitivity was quantitated as

Table 2. Individual Patients' Clinical Characteristics

	Patient Number						
	1	2	3	4	5	6	7
Age (years)	57	81	83	62	67	75	71
Duration of AV*(days)	31	34	56	3	10	10	16
$PEEP_{set}$ (cm H_2O)	5	5	11	5	5	5	5
PT-$PEEP_i$ (cm H_2O)	N/A	7	3	1	2	2	2
FT-$PEEP_i$ (cm H_2O)	N/A	8	5	2	2	2	4
C_{rs} (mL/cm H_2O)	59	56	55	77	33	40	28
R_{rs} (cm $H_2O \cdot s \cdot L^{-1}$)	20	12	21	15	12	14	25
Weaning Parameters							
V_T (mL)	200	325	300	750	300	400	350
RR (breaths/min)	28	28	35	8	37	25	26
MIP (cm H_2O)	−38	−22	−24	−36	−34	−30	−40

*AV = assisted ventilation; $PEEP_{set}$ = operator-set PEEP; PT-$PEEP_i$ = intrinsic PEEP with PT-CPAP; FT-$PEEP_i$ = intrinsic PEEP with FT-CPAP; C_{rs} = static compliance of the respiratory system; R_{rs} = total inspiratory airway resistance; V_T = tidal volume; RR = respiratory rate; and MIP = maximal negative inspiratory pressure; N/A = not available.

the airway pressure drop from the value at end exhalation that occurred at the initiation of inspiratory flow from the ventilator. WOB was calculated as the integral of negative esophageal pressure during inspiration and flow plus an estimate of chest-wall work divided by the tidal volume.[9-12] PTP was calculated as the integral of esophageal pressure and time for the duration of contraction of the respiratory muscles.[9,13,14]

The tidal volume, respiratory rate, sensitivity, WOB, and PTP were recorded and averaged for the first 10 breaths after each 15-minute test period using the printout provided by the monitor. (Preliminary calculation demonstrated no difference in results using 10 breaths versus the full 40 breaths provided by the monitor; therefore, for ease of statistical analysis, we chose to use the first 10 breaths.) The differences between the two test conditions were assessed using 2-way repeated-measures analysis of variance except for the differences in $PEEP_i$ values, which were assessed using a Wilcoxon signed ranks test. A value of $p < 0.05$ was considered statistically significant.

Results

Seven patients (3 men) whose mean (SD) age was 70.9 (9.6) years (range 62-83 years) were included in the study. Their individual, clinical characteristics are shown in Table 2. None of the patients was receiving sedation at the time of the study. The mean values (based on 10 breaths) for tidal volume, respiratory rate, and sensitivity for each of the patients during PT-CPAP and FT-CPAP are shown in Table 3. There was no statistically significant difference in any of these variables between PT-CPAP and FT-CPAP. The 10-breath mean values of WOB and PTP for each patient during PT-CPAP and FT-CPAP are given in Table 4. The mean (SD) WOB for all patients on PT-CPAP was 1.24 (0.29) J/L and for FT-CPAP was 1.19 (0.31) J/L ($p = 0.79$). Because total work depends on minute ventilation, work-rate was also calculated. The mean (SD) work-rate for patients on PT-CPAP was 12.64 (4.33) J/min and for FT-CPAP 12.05 (4.67) J/min ($p = 0.81$). The mean PTP on PT-CPAP was 282 (96) cm $H_2O \cdot s \cdot min^{-1}$ and on FT-CPAP was 251 (88) cm $H_2O \cdot s \cdot min^{-1}$ ($p = 0.78$).

Based on these 7 patients, the study had a power of more than 80% to show a 50% difference in WOB and PTP between the two test periods.

Table 3. Average (SD) Values for Tidal Volume (V_T), Respiratory Rate (RR), and Sensitivity during FT- and PT-CPAP

Variable	PT-CPAP	FT-CPAP	p
$V_{T\,(mL)}$	495 (33)	467 (30)	0.44
RR (breaths/min))	30 (10)	25 (9)	0.48
Sensitivity (cm H_2O)	4.11 (1.54)	4.41 (1.56)	0.73

Discussion

In a group of intubated patients with a history of COPD, we were unable to demonstrate a statistically significant difference in inspiratory WOB or PTP with FT-CPAP compared to PT-CPAP. Our results are similar to those from a previous bench study by Banner et al[1] who did find a significant difference between the two techniques, but the difference was small and considered clinically unimportant.

On the other hand, our results differ from previous clinical studies. Sash et al[7] reported no difference in total WOB (inspiratory and expiratory) during FT-CPAP in a study of non-intubated normal subjects breathing through endotracheal tubes, although inspiratory WOB decreased with flow-triggering. However, Sash et al used a CPAP of zero and did not specify the baseline and sensitivity flows. Similarly, Sassoon et al[5] found that the inspiratory airway pressure drop and the inspiratory WOB of 10 healthy volunteers who were not intubated were significantly less with FT-CPAP (using base flows of 5 and 20 L/min) compared to PT-CPAP using a PEEP of 5.0 or 10 cm H_2O in both test conditions.

Table 4. Individual Patient Values for Intrinsic PEEP ($PEEP_i$), Work of Breathing (WOB), and Pressure-Time Product (PTP) during PT- and FT-CPAP

	PT-CPAP			FT-CPAP		
Patient	$PEEP_i$ (cm H_2O)	WOB (J/L)	PTP (cm $H_2O \cdot s \cdot min^{-1}$)	$PEEP_i$ (cm H_2O)	WOB (J/L)	PTP (cm $H_2O \cdot s \cdot min^{-1}$)
1	N/A	0.89	217	N/A*	0.79	193
2	7	1.63	430	8	1.47	433
3	3	1.61	312	5	1.63	293
4	1	1.40	283	2	1.37	221
5	2	0.98	239	2	1.05	251
6	2	1.06	151	2	0.85	129
7	2	1.44	258	4	1.49	281

* N/A = not available.

Saito et al[4] reported that WOB and the inspiratory airway pressure drop in 8 intubated patients with unspecified diagnoses were less with FT-CPAP using a base flow of 10 L/min compared with PT-CPAP. Esophageal pressure changes were not monitored; therefore, WOB as they defined it was limited to the imposed WOB of the ventilator circuit.

Sassoon et al[6] also assessed FT-CPAP by using a base flow of 10 L/min in intubated patients with a history of COPD. In contrast to our study, these investigators found that inspiratory WOB was significantly less with FT-CPAP and continuous-flow CPAP than with PT-CPAP. In another study by Sassoon,[15] FT-CPAP was compared to T-piece or pressure support by measuring PTP in 10 intubated patients, 7 of whom had COPD. However, because no comparison was made with PT-CPAP, those results cannot be directly compared with ours.

Thus, four previous clinical studies have shown FT-CPAP to be superior to PT-CPAP in terms of WOB in both intubated and nonintubated subjects. The primary reason for the reported superiority of flow-triggering in these studies may have been improved triggering sensitivity. With the 7200ae, a little-discussed feature of flow-triggering is the 2 cm H_2O inspiratory pressure support provided, which may substantially reduce WOB.

Whereas, our findings differ from those previously reported, but our subjects and methods are different. In comparison to the one study of COPD patients,[6] our subjects were older and had more severe COPD—they had CO_2 retention, required longer duration of ventilation, and had higher values of inspiratory airway resistance and $PEEP_i$. WOB as conventionally measured requires that a volume of gas be moved, but gas movement does not occur in patients with $PEEP_i$ until the patient overcomes the $PEEP_i$. Measurement of WOB cannot begin until that point is reached. In our study, as long as the $PEEP_i$ remained stable between FT-CPAP and PT-CPAP, differences in both measured sensitivity and inspiratory WOB should have been detected regardless of the level of $PEEP_i$. In fact, $PEEP_i$ did not differ significantly between the two test conditions [mean (SD) difference = −1.0 (0.89) cm H_2O, $p = 0.068$], yet we were unable to demonstrate a difference in sensitivity (Table 3) or WOB (Table 4).

The PTP approximates total metabolic work (including work done to overcome mechanical and isometric forces). PTP may therefore be a more reliable indicator than WOB of oxygen consumed by the respiratory muscles, especially in COPD patients when $PEEP_i$ may be present.[16] Because its measurement starts at the beginning of respiratory muscle contraction, it includes work done to overcome $PEEP_i$.

Our study was done in a clinical setting in a defined group of patients who were being weaned from the ventilator. Although the study sample was small, our power values were adequate to detect a clinically important difference between the two options. However, there may be differences less than 50% that are clinically important but they would require a much larger sample to expose statistical significance.

We acknowledge two potential problems with the present study. First, we utilised FT-CPAP with a base flow of 20 L/min, and our study was completed before Kirton et al[12] suggested that lower flows might be preferable to minimize inspiratory WOB. The high base flow used in our study may have masked any benefit of FT-CPAP compared to PT-CPAP, although Sassoon et al [5] found no difference in WOB between base flows of 5 and 20 L/min. Second, we did not measure expiratory WOB. To our knowledge, no other published studies have examined expiratory WOB during FT-CPAP.

In Conclusion

Our study is only the second that has examined FT-CPAP in intubated critical care patients with COPD and the only clinical study to examine total inspiratory WOB and PTP including that done to overcome the endotracheal tube, circuit, and ventilator. We have shown that when the ventilator CPAP system functions properly, there is no reduction in inspiratory WOB or PTP with FT-CPAP with the settings and the patient population used in this study.

This raises the question of the appropriateness of developing and marketing technology without adequately studying its clinical utility in critical care patients. In the present economic climate, particularly, it is surprising to us that new options can be introduced to the market in this manner. Flow-triggering is an example of this process. Its place in treatment is unclear in spite of five postmarketing studies—yet flow-triggering is now available on a number of microprocessor ventilators.

Given that such new options are associated with increased costs, we believe that it would be more appropriate to ensure properly conducted and reported clinical testing (perhaps by contract with specific clinical test sites), before any new mode or option is introduced. This would enable respiratory care practitioners to make more informed decisions when purchasing ventilators and to better tailor the ventilator to suit the individual patient. Money might be saved.

ACKNOWLEDGMENTS

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PRODUCT SOURCES

Ventilator:
Puritan-Bennett 7200ae, Puritan-Bennet Corp, Carlsbad CA

Pulmonary Mechanics Monitor:
Model CP-100, Bicore Monitoring Systems, Irvine CA
Pneumotachograph:
Varflex, Bicore Monitoring System, Irvine CA

Esophageal Balloon Catheter:
Smart Cath, Bicore Monitoring Systems, Irvine CA

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A New Respiratory Monitor That Enables Accurate Measurement of Work of Breathing: A Validation Study

Paul B Blanch BA RRT and Michael J Banner PhD

BACKGROUND: Computerized, yet portable, bedside pulmonary monitors that can measure work of breathing (WOB) are now commercially available; however, none accurately measures WOB. The purpose of this study was to evaluate new software designed to measure WOB by means of the Campbell diagram and to test the agreement between a monitor (Model CP-100, Bicore, Irvine CA) programmed with the new software and the conventional method of measuring WOB. MATERIALS & METHODS: Using a lung model of our own devising, we compared WOB measurements between the monitor and conventional laboratory equipment. Inspiratory flowrate, tidal volume (V_T), and resistance of the model were adjusted to produce WOB ranging from 0.80 to 3.25 J/L. Regression analysis and calculation of bias and precision were performed for these data. RESULTS: For total, elastic, and resistive WOB, the two methods of measurement correlated strongly and positively. For all values of WOB, the correlation coefficients (r) and coefficients of determination (r^2) were close to 0.99 ($p < 0.0001$); bias was minimal (–0.05 J/L) and precision acceptable (0.06 J/L). CONCLUSION: Data from a lung model reveal that a respiratory monitor programmed with appropriate software can accurately measure total, elastic, and resistive WOB. The monitor may eventually prove useful for clinically assessing WOB, which then can be used to adjust the ventilator to optimize respiratory muscle loads. [Respir Care 1994;39(9):897-905.]

Background

Either pulmonary disease, failure of the respiratory pump, or both can cause ineffective gas exchange. Regardless of the cause, mechanical ventilation can maintain or support gas exchange. Furthermore, depending on the mode or level of ventilatory support, mechanical ventilation partially or totally unloads the respiratory muscles. Too much support totally unloads the muscles and, in time, they may atrophy.[1] Too little support imposes the risk of respiratory muscle fatigue.[1] In either case, the duration of mechanical ventilation may be needlessly prolonged—for reconditioning if respiratory muscles atrophy or for rest if they are chronically fatigued.[1] Therefore, optimizing the mechanical ventilator to each patient's unique needs requires information on respiratory muscle load as well as gas exchange. Work of breathing (WOB) measurements represent one approach to assessing the afterload on respiratory muscles and may provide a quantitative and goal-oriented method for setting the ventilator.

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The authors have no financial interest in any of the products mentioned in the text.

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Materials and Methods

Conventional vs Campbell-Diagram WOB Measurement

The conventional method of measuring WOB requires a trained technician and, typically, at least the following equipment: pressure transducers, multichannel polygraph recorder, flow integrator, pneumotachograph (flow sensor), X-Y plotter, and computerized planimeter. Recently, several manufacturers have incorporated all this equipment into microprocessor-controlled bedside respiratory monitors that are portable and easy to use. To our knowledge, however, none accurately measures WOB under all conditions. Most calculate WOB by measuring the area of an esophageal or airway pressure-volume (P-V) loop. Others (including early versions of the monitor evaluated here) measure work by assuming a normal chest-wall compliance (C_{cw}). At our request, Bicore Monitoring Systems developed software for their

CP-100 monitor* that determines WOB by using an established physiologic model—the Campbell diagram.[2-6] A monitor programmed with Campbell-diagram software allows the option of measuring C_{cw} before computing actual WOB. This option may be disregarded, in which case the monitor uses a default value representing normal adult C_{cw} (approximately 200 mL/cm H_2O). In practice, a monitor using such a default value underestimates WOB when C_{cw} is less than normal. Therefore, the purpose of this study was twofold—to evaluate new software designed to measure WOB when C_{cw} varies markedly from normal and to validate WOB as measured by this programmed monitor from a comparison with WOB measured using conventional laboratory equipment.

Description of Campbell Diagram

In physics, work (W) is defined as the product of force (F, measured in grams or kilograms) and distance (D, measured in centimeters or meters).

$$W = F \cdot D.$$

By the process of substitution, the equation can be transformed into a form that relates directly to the mechanics of breathing. First, because force is defined as the product of pressure (P) and area (A), $P \cdot A$, we can substitute this product for F.

$$W = P \cdot A \cdot D.$$

Next, the product of area and distance is volume (V), which can substitute for $A \cdot D$.

$$W = P \cdot V.$$

In this form, the equation describes the mechanical work needed to move a specific volume of gas. However, we cannot use this equation to determine WOB because the equation only describes work under the conditions of a specific pressure and volume; whereas during spontaneous breathing, pressure and volume vary continuously in both magnitude and direction. To account for this mathematically, by means of calculus, we use the integral of pressure relative to volume.

$$W = \int P \, dV.$$

For clinical application, rather than solving this equation, we determine the area (generally by planimetry) enclosed within the P-V loop formed when driving pressure (intraesophageal pressure, or P_{es}) is plotted against V over time. Although this equation or the measurement of area within the P-V loop comes closer than the physical definition of work ($W = F \cdot D$) to measuring the WOB, inaccuracy remains. By not including a portion of the work performed to inflate the respiratory system (Fig. 1, area to the right of the P-V loop but left of C_{cw} line), a P-V loop alone underestimates WOB. The relative magnitude of this error depends on the position of the C_{cw} line. Some commercially available pulmonary monitors attempt to correct for this error by adding a factor (based on the position of the normal C_{cw} line) to the area circumscribed by each P-V loop.

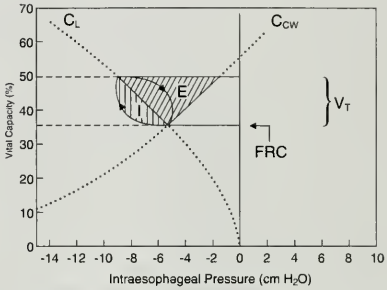

Fig. 1. A modified version of the Campbell diagram to measure the work of breathing (WOB) of a spontaneously breathing, intubated patient is devised by plotting intraesophageal pressure (P_{es}), as the surrogate of intrapleural pressure, on the horizontal axis and the change in tidal volume (V_T) labeled vital capacity, on the vertical axis. Chest-wall compliance (C_{cw}) must first be obtained by mechanically inflating the lungs of a completely relaxed patient, during which the P_{es}-V_T loop moves in a counterclockwise direction (not shown); the slope of the loop is C_{cw}, that is, the P-V relaxation curve of the chest wall. When spontaneous breathing resumes, during each inhalation (I) and subsequent exhalation (E), the same P_{es}-V_T loop moves in a clockwise direction; the slope of the loop is taken as lung compliance (C_L). Next, the stored C_{cw} line is plotted to construct a Campbell diagram for that particular patient. This line originates from the coordinates of volume and pressure representing end-exhalation. Under normal conditions, functional residual capacity occurs at the intersection of the two compliance lines. Physiologic resistive work and imposed work are represented by the inspiratory portion of the P-V loop (▥). Elastic work is the triangular area subtended by the two compliance lines(▨). Total measured WOB is the resistive (physiologic + imposed) + elastic work.

Campbell first recognized these difficulties when he attempted to calculate WOB by using only the intraesophageal P-V loop or the more complex Rahn diagram.[7] To resolve the problems, he developed a graphic approach to measuring WOB (Fig. 1).[7] The first step in this approach is to directly measure C_{cw} (which remains relatively constant for reasonable periods of time). A practical and reliable way to do this involves mechanically inflating the lungs and thorax of a completely relaxed or paralyzed patient. Under these conditions, C_{cw} is taken as the slope of the P-V loop, which represents the change in tidal volume (V_T) relative to the change in intrapleural pressure (P_{pl}).

*Manufacturers and suppliers are identified in the Product Sources section at the end of the text.

Thus,

$$C_{cw} = \frac{V_T}{P_{pl(final)} - P_{pl(initial)}}$$

Instead of measuring P_{pl} directly, pressure readings from a properly positioned esophageal balloon (P_{es}) accurately reflect P_{pl} and may be used as its surrogate.[8] After C_{cw} has been determined, P-V (P_{es}-V_T) loops of representative spontaneous breaths are obtained. Measured C_{cw} and P-V loops are then combined into a single, composite graph, and WOB measured from it by planimetry (Fig. 1).

Physiologic resistive WOB is the work performed by the respiratory muscles to overcome the resistance (R) of the airways and tissues to gas flow.[9] During spontaneous breathing through an endotracheal tube (ETT) connected to a ventilator, triggering the ventilator and breathing across the ventilator circuit and through the ETT superimposes an additional resistive work load (imposed work [WOB_{imp}]) on the respiratory muscles.[10,11] The measured resistive WOB (WOB_R = physiologic + imposed) is represented by the area within the inspiratory portion of the P-V loop (Fig. 1). Elastic WOB (WOB_{el}) is the work of respiratory muscles to overcome the elastic forces of the respiratory system (lungs and chest wall) during inflation (Fig. 1).[9] WOB_{el} is represented graphically by the area subtended by the two compliance lines (C_{cw} and compliance of the lung, or C_L). At this point, the position of the C_{cw} line plays an essential role in the measurement of WOB_{el} (Fig. 2).[12] An improperly positioned C_{cw} line may result in a significant underestimate of both WOB_{el} and total WOB (WOB_{tot}). Moreover, based on experience in our intensive care units, normal C_{cw} seems to be the exception, rather than the rule. Among 30 patients, mean (SD) C_{cw} was approximately 109 (37) mL/cm H_2O, with values ranging from 50 to 180 mL/cm H_2O.[13] This work has been corroborated at another medical center (JM Civetta, personal communication, 1994). WOB_{tot} is the arithmetic sum of the resistive and elastic work components.

$$WOB_{tot} = WOB_R + WOB_{el}.$$

Evaluation Methods

All work values (WOB_{tot}, WOB_R, and WOB_{el}) were determined by using the Campbell diagram.[7,14] The respiratory monitor used a computer algorithm and displayed all WOB values breath by breath. Simultaneously, an individual P-V loop was obtained for each breath by using conventional laboratory equipment. Later, each of these loops was transformed by hand into a Campbell diagram, measured by computerized planimetry, and converted into appropriate work units (J/L).

Lung Model. Commercially available lung models neither incorporate a structure that simulates the chest wall nor

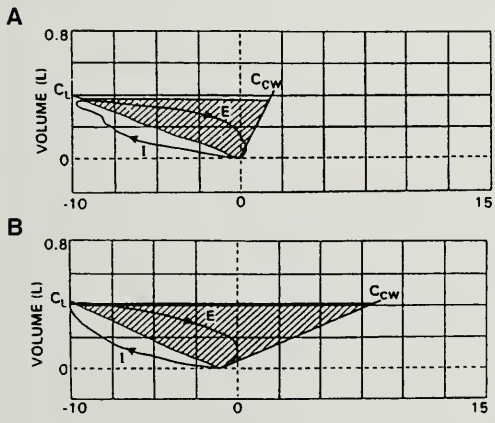

Fig. 2. Examples of Campbell diagrams showing an increase in elastic work of breathing (WOB_{el}) from decreased chest-wall compliance (C_{cw}) as a result of ascites. The Campbell diagram display from the monitor plots volume over intraesophageal pressure (P_{es}) during spontaneous inhalation (I) and exhalation (E). The P-V loop moves in a clockwise direction; the slope of the loop represents lung compliance (C_L). The C_{cw} was obtained previously (Fig. 1). In this example, before ascites develops (A), WOB_R is 0.22 J/L and total work of breathing (WOB_{tot} = elastic work + resistive work) is 0.74 J/L. The C_{cw} is 180 mL/cm H_2O. When severe ascites follows (B), the abdomen becomes markedly distended, which decreases C_{cw} to 53 mL/cm H_2O. Note the nearly identical size and shape of the two P-V loops. The WOB_{el}, however, increases from 0.52 to 1.0 J/L, as indicated by the triangular-shaped areas (▨) subtended by the compliance curves. Had a normal C_{cw} been assumed, after the onset of ascites, the measured WOB_{tot} would have been an underestimate by more than 40%. WOB_R is resistive work of breathing. (Adapted from Reference 12, with permission.)

provide an appropriate location from which to monitor P_{pl}. Therefore, we devised a model of the respiratory system (Fig. 3). The model consisted of two spring-loaded bellows, a plastic canister, a series of valves actuated by a pressure sensor, and a piston pump. One bellows (simulating the lung) was suspended within the plastic canister (simulating the chest wall) and the other was attached to the outside of the canister (Fig. 3). An opening in the side of the canister was connected by a series of valves to a reciprocating piston pump. This type of pump was selected because it generates a physiologic, sinusoidal inspiratory flow waveform.

The downstroke of the piston decreased pressure, thus mimicking P_{pl} changes during spontaneous inhalation and inflating the encased bellows (lung). Normally, respiratory muscle activity ceases soon after each spontaneous inhalation has been completed, and exhalation is passive. To avoid an unnatural, active exhalation phase in the lung model, we devised a series of valves actuated by a pressure sensor. This system served to sense and then isolate (disconnect) the lung model from the piston during the exhalation phase. Once isolated from the piston, the bellows (lung) exhaled passively in response to its own elastic recoil, rather than from positive pressure generated by the piston's changing direction (up stroke, Fig. 4). At the onset of inhalation, the same sensor detected the change in piston direction and reconnected the pump and lung, which enabled the piston to decrease pressure within the canister and, thus, to simulate an active inhalation.

An endotracheal tube (ETT) was placed into the simulated trachea of the lung model (Fig. 3). Airway pressure (P_{aw}) was measured through a fitting attached directly to the ETT and connected to a pressure transducer. The P_{pl} was measured through a port in the side wall of the plastic canister (chest wall), and the P_{pl} signal was simultaneously directed to a second pressure transducer and to the respiratory monitor. Changes in flowrate and V_T were measured by a standard pneumotachograph and with a miniature pneumotachograph from the respiratory monitor. The pneumotachographs were attached, in series, directly to the ETT (Fig. 3). Conventional pressure, volume, and flow signals were directed to an amplifier-integrator-polygraph recorder system. From the polygraph, P_{pl} and V_T signals were then directed to an X-Y plotter, which recorded the P_{pl}-V_T loops onto graph paper. Surface areas of the regions defined as WOB_R and WOB_{el} on each Campbell diagram were measured by a computerized system with an electronic planimeter. Later, the measured surface areas were converted into J/L. This step-by-step conversion is given in Appendix 1. Using a computer algorithm, signals from the miniature pneumotachograph, and its internal P_{pl} transducer, the respiratory monitor simultaneously calculated WOB_{tot}, WOB_R, and WOB_{el} and displayed each with a representative Campbell diagram for each studied breath (Fig. 3). Conventional laboratory equipment was calibrated using a 3-L calibration syringe and a water manometer.

Before WOB was determined by either method, C_{cw} and C_L of the lung model were determined. The monitor pro-

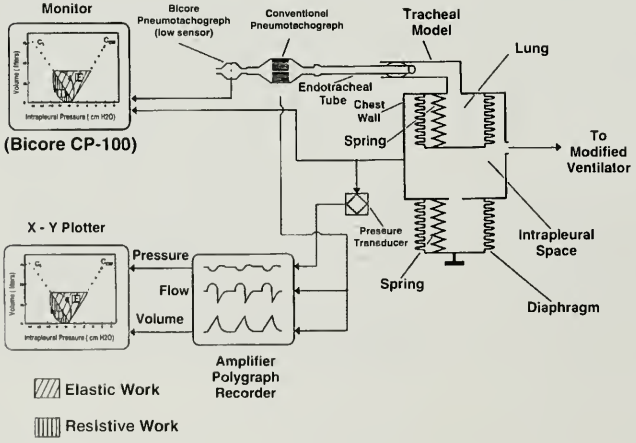

Fig. 3. Model of the respiratory system used for simulating spontaneous breathing and simultaneously measuring the work of breathing (WOB). A spring-loaded bellows ('lung'), sealed in a plastic canister ('chest wall'), simulates the respiratory system. A piston pump, mimicking the action of respiratory muscle contraction, intermittently decreases pressure within the canister and simulates changes in 'intrapleural pressure.' Similarly, decreases in intrapleural pressure changes can be produced manually by pulling down a second spring-loaded bellows ('diaphragm'), also attached to the canister. The conventional pneumotachograph measures volume and flow, and a pressure transducer measures simulated intrapleural pressure.

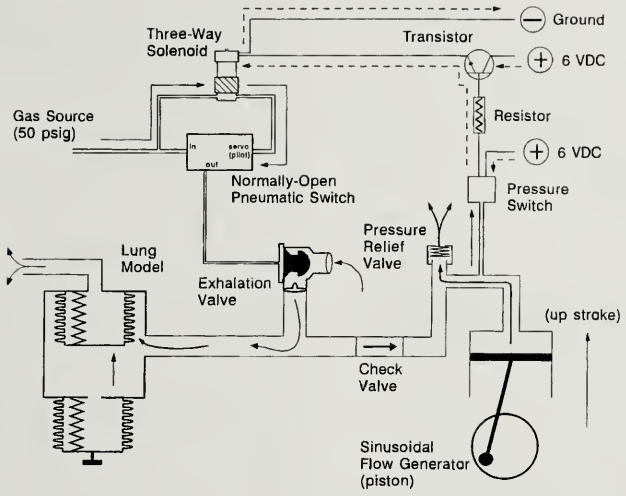

Fig. 4. A schema of the system used to isolate the piston pump (sinusoidal flow generator) from the lung model during exhalation. At the moment the piston begins moving upward, positive pressure builds between the check valve, the pressure-relief valve, and the pressure switch. When the increasing pressure reaches approximately 10 cm H_2O, the pressure-relief valve opens and maintains this pressure while venting the remainder of the gas displaced by the piston (solid arrows). Immediately prior to this, at approximately 2cm H_2O pressure, the pressure switch senses positive pressure, completes a circuit and allows 6-volt DC (6 VDC) to flow through the resistor (dashed arrows). The electrified resistor, in turn, allows current to flow through the transistor, which actuates the three-way solenoid (dashed arrows). Once energized, the solenoid allows gas at 50 pounds per square inch gauge (psig) to flow through and turn off the normally open pneumatic switch (solid arrows). Without a pressure signal from the normally open switch, the exhalation valve opens, the lung model exhales passively (solid arrows), and gas evacuated from the canister during inhalation returns via the open exhalation valve (solid arrows). At the onset of inhalation (downstroke of the piston), the absence of positive pressure closes the pressure relief and switch, breaks the electric circuit and closes the three-way solenoid (not shown). Without a signal from the three-way solenoid, the normally open switch reopens, closes the exhalation valve, and reconnects the piston to the canister of the lung model (also not shown).

gram contains an algorithm specifically designed for measuring C_{cw} and storing those data electronically. The values measured by the monitor were virtually identical to those obtained by the conventional method. With the calibration syringe, five consecutive 100-mL increments of gas were injected through the pneumotachographs and into the simulated lung. When a static pressure had been reached after each 100-mL injection, the total injected volume relative to the changes in P_{pl} and P_{aw} was recorded. This process, which simulated the mechanical inflation of a completely relaxed patient, allowed calculation of C_{cw} (Eq. 1) and compliance of the respiratory system (C_{rs}), defined as the slope of the P-V loop of airway pressure and V_T.

$$C_{rs} = \frac{V_T}{P_{aw(final)} - P_{aw(initial)}}$$

After C_{rs} was determined, the C_L of the lung model was calculated mathematically.

$$\frac{1}{C_L} = \frac{1}{C_{rs}} - \frac{1}{C_{cw}}.$$

For the model, C_{cw} was 0.043 L/cm H_2O and C_L was 0.046 L/cm H_2O. ETTs with an internal diameter from 4.0 mm through 8.0 mm (in 1-mm increments) were in turn inserted into the trachea of the lung model to simulate changes in resistance. The piston pump was adjusted to, and maintained at, a frequency of approximately 10 breaths/min. By adjusting the velocity and distance traveled by the piston, the peak inspiratory flow demand ranged from 15 to 75 L/min and V_T from 200 to 700 mL. At each of 30 different combinations of ETT, resistance, peak inspiratory flow demand, and V_T, WOB was measured for three representative breaths.

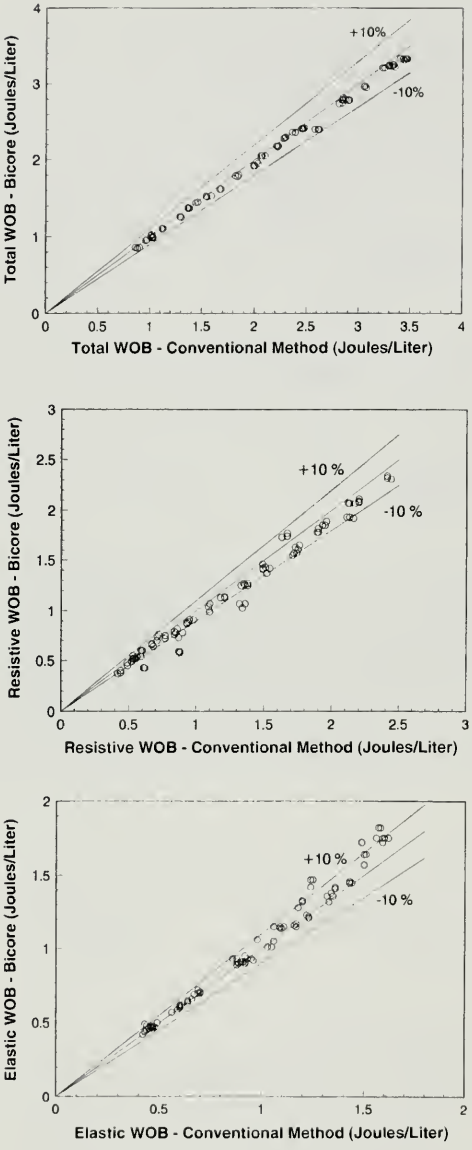

Fig. 5. Total (top), resistive (middle), and elastic (bottom) work of breathing as measured by a bedside pulmonary monitor plotted against those measured by conventional methods. The center line is the line of identity; lines representing ±10% from the conventional measurements are indicated.

WOB_{tot}, WOB_R, and WOB_{el} measured by conventional means were compared graphically to those reported by the respiratory monitor. For each pair of measurements, regression analysis was performed, and bias and precision were calculated. Bias was taken as the difference between the conventional and the displayed work value. For WOB, measured bias was plotted as a function of the average value obtained by both conventional means and the programmed monitor.[15]

Evaluation Results

The WOB_{tot} reported by the monitor agreed with conventional measurements (±10%) (Fig. 5, top). Paired measurements for WOB_R and WOB_{el} were scattered more widely (less precise) but, with a few exceptions, also agreed (±10%). As the WOB_{tot} progressively increased, however, the WOB_R reported by the monitor tended to be slightly lower than that obtained by the conventional method (Fig. 5, middle). Similarly, the monitor WOB_{el} tended to be sightly higher than conventional WOB_{el} (Fig. 5, bottom). Although not clinically important, a slight bias of the monitor occurred in each of the three measurements. For WOB_{tot} (Fig. 6), average bias was approximately −0.05 J/L, for WOB_R −0.07 J/L, and for WOB_{el} 0.03 J/L. The 95% confidence intervals (precision) for each measured parameter were also within clinically acceptable limits: for WOB_{tot} (Fig. 6) approximately ±0.06 J/L, for WOB_R ±0.12 J/L, and for WOB_{el} ±0.08 J/L.

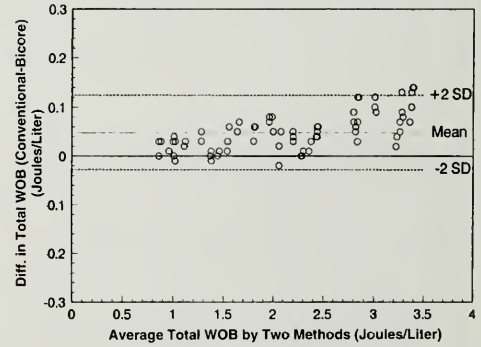

Fig. 6. Analysis of the limits of agreement in total work of breathing (WOB) measured by conventional means minus that measured by the programmed pulmonary monitor plotted on the Y-axis. The mean of the arithmetic sum of conventional measurement and monitor reading is plotted on the X-axis. The mean difference (−0.05 J/L), or bias, is shown; lines representing ±2 standard deviations (SD) from the mean difference (precision = ±0.06 J/L) are also indicated.

Regression analysis revealed that correlation coefficients (r) and coefficients of determination (r^2) were close to 0.99 and highly significant ($p < 0.0001$) for WOB_{tot}, WOB_R, and WOB_{el}.

Discussion

Work of breathing could be a clinically important and therapeutically useful indicator. Despite its potential, however, technical impediments have limited clinical applications until the advent of portable, microprocessor-controlled pulmonary monitors. Our study demonstrates that such a monitor, programmed with appropriate software (Campbell diagram), can accurately and reliably measure WOB, even when C_{cw} varies markedly from normal.

In this study, minor disagreements in measured WOB_R and WOB_{el} likely resulted in part from placing the two pneumotachographs in series (Fig. 3), which allowed for simultaneous measurements of WOB with both systems but concomitantly caused the second pneumotachograph (conventional) to sense a larger pressure change with any particular flowrate. As a result, conventional values for WOB_R were slightly higher than those simultaneously determined by the monitor, particularly at higher inspiratory flowrates (higher WOB_{tot}).

Additional errors in partitioning work into elastic and resistive components might have occurred when the monitor plotted the C_L curve. Although the C_L of the lung model remained constant, the monitor measures C_L and plots a curve for it dynamically by connecting points of no flow (which occur at end-inhalation and end-exhalation) through each P-V loop. Under conditions of high airway resistance, flowrate may decelerate to a level below that which the monitor senses (approximately 1 to 2 L/min), before the end of inhalation or exhalation. In this situation, the C_L line plotted by the monitor varies from that plotted by the conventional technique: Placement of the C_L line partitions the WOB_{tot} area and any variation in position results in differences in the elastic and resistive components. Nevertheless, the additional scattering and errors were minor and probably not clinically important. Further, partitioning of WOB load into constituent components is of interest primarily in differentiating the various causes of respiratory muscle loading.

As mentioned, the Campbell technique requires both a properly positioned esophageal balloon and a carefully measured C_{cw}. When properly positioned, the balloon rests in the mid-to-lower third of the esophagus.[8,16] This position can be verified with the airway occlusion technique.[16] Accuracy in measuring C_{cw} requires complete relaxation of the respiratory muscles and a mechanically ventilated patient. Our approach to achieve transient respiratory muscle relaxation for adults is to administer midazolam, 1 to 2 mg.[13] During the relaxation, the ventilator rate should be increased (if necessary) to approximately 10 to 12 breaths/min. During each mechanical inflation under these conditions, esophageal pressure increases. The monitor integrates the change in esophageal pressure relative to V_T and produces a pressure-volume loop, the slope of which is taken as C_{cw}. Compliance values are then averaged and stored in the monitor's computer memory. Next, flumazenil, approximately 0.2 mg, is given to reverse the effects of the midazolam, which allows the patient to resume spontaneous ventilation.[13]

Initially, the additional hardware and procedures required to measure WOB by the Campbell diagram may seem cumbersome. Simpler inferences of work, however, such as from respiratory rate and breathing pattern, may be inaccurate and misleading.[13,17] Basing therapy on data so derived may result in severe respiratory muscle fatigue or atrophy. In either case, discontinuation of mechanical ventilation may be needlessly delayed.

In contrast to traditional approaches that rely on surrogate measurements of WOB, the effect of afterload on respiratory muscles, assessed from WOB_{tot} measurements, offers a quantitative, repeatable, and goal-oriented approach to partially or totally unloading the respiratory muscles.[13,18,19] With partial unloading, pressure support ventilation and respiratory muscles share the WOB. A reasonable starting point for this approach might be to allow the respiratory muscles to perform normal WOB (approximately 0.3 to 0.6 J/L)[13,19] and to set pressure support ventilation to provide the remainder of the work. In some cases, however, respiratory muscle atrophy, weakness, or fatigue prevents the patient from tolerating even normal WOB. The load tolerance for some patients may vary. In these instances, the monitor allows the clinician to first quantify and then manipulate WOB_{tot} to produce a tolerable load. In theory, maintaining a normal or tolerable load throughout ventilatory support would preserve respiratory muscle conditioning. Alternatively, a clinician using the monitor may choose to increase the level of pressure support ventilation until the measured WOB_{tot} equals zero. This strategy provides complete respiratory muscle rest. Many authorities now contend that chronically fatigued respiratory muscles recover when allowed to rest for at least 24 hours.[20-22]

For debilitated or ventilator-dependent patients, the monitor allows a periodic but quantitative application of additional work (exercise). Exercise periods help to recondition the respiratory muscles by gradually building both strength and endurance.[21] When a patient's condition and stamina improve to the point that a load comparable to that required after endotracheal extubation is maintained, presumably extubation will be successful.

Conclusion

As discussed, real-time, bedside measurement of WOB_{tot} represents one possible approach to managing WOB (respiratory muscle afterload) during ventilatory support. The clinical use of this measurement, however, is predicated on reliability and accuracy, which, in turn, requires knowledge of how to correctly measure the resistive and elastic components of WOB. In addition, because a distinct possibility exists that C_{cw} might be significantly lower than normal, C_{cw} must be measured frequently to ensure accurate WOB measurements. The Campbell diagram provides a practical mechanism for incorporating C_{cw} into bedside measurements of WOB. Our data reveal that a monitor programmed with Campbell-diagram software provides an easy-to-use, accurate, and reliable method of measuring WOB.

PRODUCT SOURCES

Respiratory Monitor:
CP-100, Bicore, Irvine CA
Software for CP-100, Bicore, Irvine CA

Piston Pump:
JH Emerson Co, Cambridge MA

Endotracheal Tubes:
Sheridan, Argyle NY

Pressure Transducers:
Model P7 & PM15E, Gould-Statham, Oxnard CA

Pneumotachograph:
Fleisch No. 2, Blue Bell PA

Polygraph:
Model 7B, Grass Medical Instruments, Quincy MA

X-Y Plotter:
Model 7035B, Hewlett-Packard, San Diego CA

Computer:
PC Microcomputer, Model E15A - VLB 486-66DX2,
COMTRADE, City of Industry CA

Electronic Planimeter:
Model 9111A, Graphics Tablet, Hewlett-Packard, San Diego CA

3-L Calibration Syringe:
WE Collins, Braintree MA

Water Manometer:
Westcor Inc, Logan UT

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APPENDIX

Determination of a Quantity of Work from Surface Area (in mm^2) Taken from a Plot on Graph Paper

1. The distance traveled by the plotter pen with each cm H_2O pressure change is first measured (for example: P_{dist} = 1 cm H_2O/mm).

2. The distance traveled by the plotter pen with each mL of V_T is then measured (for example: V_{dist} = 50 mL/mm).

3. In our example, the P-V relationship for a mm^2 becomes
$$(P_{dist}) \cdot (V_{dist}) = (1 \text{ cm } H_2O/mm) \cdot (50 \text{ mL/mm})$$
$$= 50 \text{ cm } H_2O \cdot mL/mm^2.$$

4. The conversion factor for cm $H_2O \cdot mL$ into $kg \cdot m$ is
$$1 \text{ cm } H_2O \cdot mL = 10^{-5} \text{ kg} \cdot m.$$

5. Each cm $H_2O \cdot mL/mm^2$ is now converted to $kg \cdot m/mm^2$.
$$(50 \text{ cm } H_2O \cdot mL/mm^2) \cdot (10^{-5} \text{ kg} \cdot m/\text{cm } H_2O \cdot mL)$$
$$= 5 \cdot 10^{-4} \text{ kg} \cdot m/mm^2.$$

6. The conversion factor for $kg \cdot m$ into joules
$$1 \text{ kg} \cdot m = 9.81 \text{ J/kg} \cdot m.$$

7. Each $kg \cdot m \cdot mm^{-2}$ is thereby converted to J/mm^2
$$(9.81 \text{ J/kg} \cdot m) \cdot (5 \cdot 10^{-4} \text{ kg} \cdot m \cdot mm^{-2})$$
$$= 4.90 \cdot 10^{-2} \text{ J/mm}^2.$$

8. This derived conversion factor ($4.90 \cdot 10^{-2}$ J/mm^2) allows surface area measured in mm^2 to be directly converted into joules.

9. To obtain work in J/L, the total number of joules of work for each breath is then divided by the V_T of that specific breath.

10. Using our example with V_T = 500 mL, measured surface area = 25 mm^2,
$$\text{work} = (25 \text{ mm}^2) \cdot (4.9 \cdot 10^{-2} \text{ J/mm}^2) = 1.22 \text{ J}$$
and
$$\text{work (J/L)} = \frac{1.22 \text{ J}}{0.5 \text{ L}} = 2.44 \text{ J/L}.$$

Unilateral Lung Disease Treated with Patient-Triggered Independent-Lung Ventilation: A Case Report

Hubert J Schmitt MD, Harald Mang MD, and Max Kirmse MD

Independent lung ventilation (ILV) has been used successfully in the operating room during thoracic surgery. However its application in critical care medicine remains controversial. A 40-year-old man with alcoholism-induced acute pancreatitis developed sepsis, left-lung consolidation, and respiratory failure that was refractory to conventional ventilation techniques. A double-lumen tracheostomy tube was placed in preparation for ILV. Two ventilators were used in the assist-control mode permitting the patient's inspiration to trigger both machines simultaneously while allowing different expiratory times dictated by the individual lung's time constant. Suctioning became increasingly productive, clearing the lung consolidation, and in 2 days the patient was placed back on a single conventional ventilator. In less than 2 weeks, the patient had recovered and was discharged from the hospital. [Respir Care 1994;39(9):906-911]

Introduction

Independent lung ventilation (ILV) with positive end-expiratory pressure (PEEP) via a double-lumen endotracheal tube (Fig. 1) has been used in the operating room during thoracic surgery. Although some reports have shown a beneficial effect for patients in acute respiratory failure caused by a unilateral lung infection,[1-3] the application of ILV in critical care medicine remains controversial.[4]

The indications for the application of ILV include severe unilateral bronchopleural fistulas and inhomogenous lung disease when conventional ventilatory support is not successful. Several authors[1-4] report a dramatic improvement in gas exchange during ILV, which may have been life-saving in some patients. Several techniques for applying ILV have been established: one ventilator with separate circuits and exhalation valves[5-7] or two ventilators operating synchronously[8,9] or asynchronously.[10,11] Synchronous ILV (SILV) with a master and a slave ventilator[12] is rarely used because it requires special equipment.

We report a patient with unilateral atelectasis and pneumonia causing hypoxemia unresponsive to prolonged conventional ventilatory support with PEEP. ILV was established using two nonsynchronized ventilators operating in the assist/control mode. Although the ventilators were not synchronized by design, they were functionally synchronous because the patient was able to trigger both ventilators simultaneously.

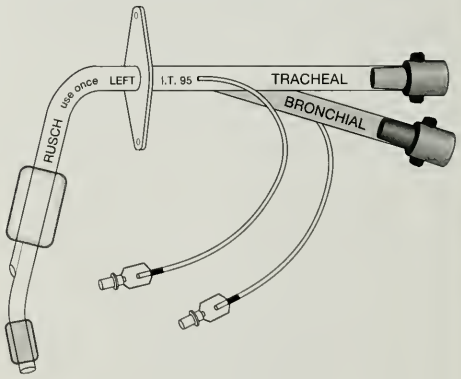

Fig. 1. Double-lumen tracheostomy tube, Tracheopart 39-Fr, 85-mm. The lumen with the small distal cuff inserts into the main bronchus—in our case, the left main bronchus—completely isolating one lung from the other.

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None of the authors has a financial interest in any products mentioned in the text.

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Case Report

A 40-year-old Caucasian man with a history of alcohol abuse and acute pancreatitis was admitted to the hospital for drainage of a chronic pseudocyst. He was transferred postoperatively to the surgical intensive care unit where he developed sepsis syndrome followed by acute pulmonary failure. The patient showed signs of moderate respiratory distress with a respiratory rate between 30 and 40 breaths/minute, a temperature of 38.9°C, and a disoriented mental state. Blood pressure was 140/70 mm Hg [18.7/9.3 kPa], and heart rate 175 beats/minute. Laboratory testing revealed a leukocytosis (WBC = 22 × 10^9/L) and thrombocytopenia (RBC = 70 × 10^9/L). Blood cultures were drawn and ofloxacin was begun (200 mg I.V. twice daily). An arterial blood gas sample (ABG) showed pH 7.37, P_{aO_2} 62 torr [8.3 kPa], and P_{aCO_2} 28 torr [3.7 kPa], while breathing 6 L/min oxygen via mask. Because of the development of progressive hypoxemia and impairment of consciousness, he was intubated the following day with a 9.0-mm-ID endotracheal tube and mechanically ventilated using a Servo 900C ventilator.*

Initial settings were tidal volume (V_T) 660 mL, SIMV rate 12/min, PEEP 4 cm H_2O [0.4 kPa], and F_{IO_2} 0.6. For spontaneous breaths, inspiratory pressure support was set to 14 cm H_2O [1.4 kPa]. Because oxygenation worsened, the ventilatory mode was changed to pressure-control. Ventilator settings were inspiratory pressure level above PEEP 18 cm H_2O [1.8 kPa], PEEP 10 cm H_2O [1.0 kPa], respiratory rate 16 breaths/minute, inspiratory-to-expiratory ratio (I:E) 1:1, and F_{IO_2} 0.6. The patient was sedated with midazolam (0.2 mg · kg^{-1} · h^{-1}) and sufentanil (1 μg · kg^{-1} · h^{-1}). The chest radiograph revealed bilateral lower lung opacities consistent with pneumonia and an additional atelectasis on the left side. A bronchoscopy was performed, and a small amount of mucus was removed. Cultures did not show growth of microorganisms. However, cultures obtained from the wound drainage showed growth of *Streptococcus* species, anaerobic bacteria, and fungi. Therefore, the antimicrobial regimen was changed to penicillin G, imipenem/cilastatin (Zienam), and amphotericin B.

By the end of the first week, the patient's oxygenation had deteriorated, and the chest radiograph revealed an almost complete opacity of the left lung. However, bronchoscopy did not reveal unusual amounts of mucus. In order to maintain adequate oxygenation, the ventilator was set to an inspiratory pressure level of 24 cm H_2O [2.4 kPa] above PEEP, PEEP 14 cm H_2O [1.4 kPa], respiratory rate 20 breaths/min, and F_{IO_2} 0.7. Computerized tomography (CT) showed a subhepatic abscess that was drained surgically the same day. The patient's body temperature decreased to normal values over the next 4 days. In order to reduce peak inspiratory pressures, the Servo 900C ventilator was replaced by a Servo 300 operated in the pressure-regulated volume-control mode. The ventilator settings were V_T 600 mL, PEEP 8 cm H_2O [0.8 kPa], I:E 1:1, and F_{IO_2} 0.7. The flow-trigger was activated and respiratory rates ranged from 17 to 25 breaths/minute. ABG values were P_{aO_2} 79 torr [7.9 kPa], P_{aCO_2} 52 torr [5.2 kPa], and pH 7.36. By regularly turning the patient to the right lateral position, we were able to reduce the F_{IO_2} to 0.5 in the course of the next week. A representative ABG taken near the end of the second week showed a P_{aO_2} of 76 torr [7.6 kPa], P_{aCO_2} of 51 torr [5.1 kPa], and pH 7.34.

On the 15th day of mechanical ventilation, a CT scan of the chest showed an unchanged left lung opacity (Fig. 2A). A tracheotomy was performed on the same day.

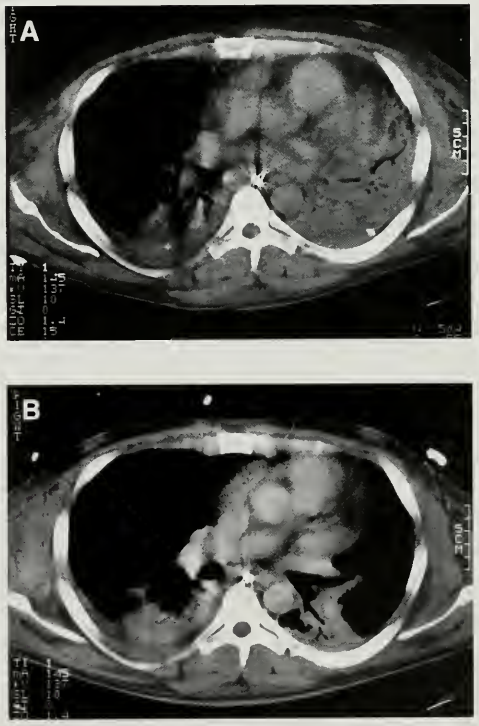

Fig. 2. (A) Transverse CT scan of the thorax before ILV. The left lung is almost entirely collapsed. (B) Corresponding CT scan after the first day of ILV. Note the resolution of the dense area in the left lung.

*Manufacturers and Suppliers are identified in the Product Sources section at the end of the text.

The patient was intubated with a left-side double-lumen tracheostomy tube (Fig. 1). The correct position of the tube was verified by fiberoptic bronchoscopy. ILV was established by two Servo 300 ventilators operating in the flow-triggered, pressure-regulated, volume-control mode. The flow-trigger enabled the patient to trigger both ventilators simultaneously. The decelerating flow pattern of the pressure-regulated volume-control mode was used to keep airway pressures as low as possible. At this time, the patient was sedated with sufentanil only. However, when respiratory rate exceeded 25 breaths/minute, the sufentanil dose was increased to 3 $\mu g \cdot kg^{-1} \cdot h^{-1}$. Initially, V_T was 300 mL for the left lung and 410 mL for the right lung. PEEP was 8.0 cm H_2O [0.8 kPa] for both lungs first, and then increased to 18 cm H_2O [1.8 kPa] for the left lung. PEEP levels were set for both lungs individually and were identical to the pressures at the inflection points of the airway pressure-volume loops obtained from the ventilator monitors. Measured ventilatory variables are summarized in Table 1. Within hours, bronchial suctioning removed large amounts of mucus from the left lung.

Gas exchange improved (F_{IO_2} decreased from 0.5 to 0.3), and the chest radiograph and the CT scan showed an almost complete clearing of the left lung opacity (Fig. 2B). After 2 days of ILV, the patient showed a sustained improvement in oxygenation and a single-lumen tracheostomy tube was placed. Due to a slow recovery from the underlying disease process (necrotizing pancreatitis), the patient was on partial ventilatory support for another week. After a total of 24 days of mechanical ventilation, the patient was weaned successfully and extubated. He was discharged from the hospital 1 week after extubation without further complications.

Discussion

In patients with predominantly unilateral lung pathology, the mechanical properties of the lungs are inhomogenous. Decreased functional residual capacity and lung compliance and increased airway resistance of the affected lung lead to a maldistribution of ventilation, with atelectasis and overdistention.[13] Applied PEEP might worsen this effect by further distending the more compliant healthy lung, resulting in increased pulmonary vascular resistance and greater ventilation-perfusion mismatch. Vascular resistance in the affected lung is also increased because of hypoxic pulmonary vasoconstriction.[14] However, the elevated alveolar pressure in the healthy lung is the primary factor that causes blood flow to shift to the less compliant lung, thus increasing intrapulmonary shunt.[15]

To counteract this effect, some authors have proposed the lateral decubitus position, the so called "good lung down" approach to increase blood flow to the healthy lung.[16,17] However, by the end of 1 week, this procedure had not resulted in a significant improvement in our patient. The failure of this approach together with the radiographic evidence of unilateral lung opacity led us to try ILV.[18] Although additional data like static compliance curves can be helpful with such decisions,[8] we considered the institution of ILV justified by the patient's clinical course alone. During ILV, the PEEP level in each lung was determined by the inflection point in the individual pressure-volume loop.[19]

The immediate improvement in gas exchange in our patient during ILV might have been due to a selective increase in mean-airway pressure ($\bar{P}_{aw}$) caused by PEEP in the affected left lung (Fig. 3).[19] The CT scan taken on Day

Table 1. Ventilator Settings and Arterial Blood Gas Values during Independent-Lung Ventilation (ILV)

	−2 hours (Before ILV)	0 hour		8 hours		16 hours		24 hours		32 hours		40 hours		44 hours (After ILV)
		Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right	
F_{IO_2}	0.5	0.5		0.4		0.4		0.3		0.4		0.3		0.4
Breaths/min	18	21		20		19		18		18		18		18
V_T (mL)	564	300	410	290	430	330	460	350	480	340	400	260	450	560
$\dot{V}_E$ (L/min)	10.5	6.7	9.1	6.0	8.8	6.5	8.7	6.5	9.0	6.4	7.4	5.3	8.3	10.5
P_{peak} (cm H_2O)	30	40	28	34	30	37	30	37	30	38	26	35	28	26
$\bar{P}_{aw}$ (cm H_2O)	18	30	17	25	18	28	18	27	19	28	18	27	17	16
PEEP (cm H_2O)	8	8	8	17	7	18	8	18	8	18	8	18	8	8
P_{aO_2} (torr)	76	95		88		88		77		117		88		78
P_{aCO_2} (torr)	51	35		35		34		35		38		40		36
pH	7.34	7.39		7.42		7.44		7.42		7.39		7.40		7.41

Left = left lung and Right = right lung. V_T = tidal volume, $\dot{V}_E$ = minute ventilation, P_{peak} = peak inspiratory pressure, $\bar{P}_{aw}$ = mean airway pressure, and PEEP = positive end-expiratory pressure.

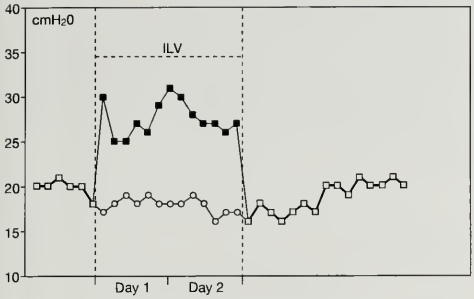

Fig. 3. Mean airway pressure before, during, and after ILV. The legend is(—○—) right lung, (—■—) left lung, and (—□—) both lungs.

l of ILV showed an almost complete resolution of atelectasis in the left lung, indicating that recruitment of collapsed alveoli had taken place (Fig. 2B). This re-expansion would also explain the rapid improvement in oxygenation. Because these data are anecdotal, one might argue that the benefit we attribute to ILV may also have occurred spontaneously or as a result of the initial therapy or natural resolution of disease. However, the prompt resolution of the lung opacity is strongly supportive of a direct and causal relationship with ILV. Our patient's response is consistent with the experience of others[6,19,20] who report that most improvements have been achieved in the first few hours of ILV. Therefore, ILV may be the technique of choice to selectively recruit a unilaterally diseased lung.[21,22] Conventional ventilation does not achieve equivalent results in unilateral lung disease because ventilation is diverted to the healthy lung, causing overdistention with an increased risk of barotrauma.[23] In addition, the reduced PEEP level in the healthy right lung causes a decrease in

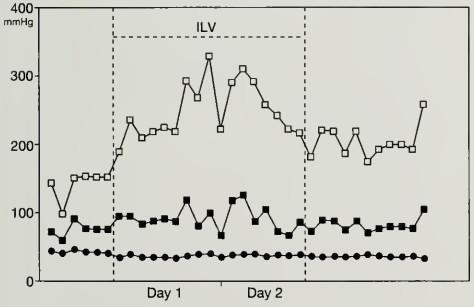

Fig. 4. Arterial oxygen (—■—), carbon dioxide (—●—) tensions, and the arterial oxygen tension/inspired oxygen fraction (—□—) before, during, and after ILV.

pulmonary vascular resistance and increases perfusion to ventilated lung.[24] In our patient, ILV led to a better matching of ventilation and perfusion, which improved gas exchange as shown by the increased P_{aO_2}/F_{IO_2} (Fig. 4).

Improved ventilation of the diseased lung allowed for more effective removal of secretions from consolidated areas and prevented mucus plugs and absorption atelectasis. This became obvious by the increased demand for suctioning in our patient. A further advantage of the double-lumen tube is that it prevents the spilling of infected secretions into the healthy lung.[25] However, the small cross-sectional area of each lumen increases airway resistance and the likelihood of obstruction by secretions. Periodic airway care and suctioning are necessary also to avoid airway obstruction that would prevent the patient from triggering both ventilators.

Similar to Chiaranda et al,[26] we measured the flow-resistive properties of the double-lumen tracheostomy tube. The resistance of the bronchial lumen (Fig. 5A) is slightly lower than that of the tracheal lumen (Fig. 5B), and both resistances are lower in the double-lumen tracheostomy tube than those in the Carlens and left-side Robertshaw tube of the same French size. In addition, we measured the work of breathing imposed by each lumen of the tracheostomy tube and found that the additional work of breathing through each lumen is similar to that of a 6.5-mm-ID endotracheal tube. Airway management problems and technical difficulties greatly contribute to the infrequent use of ILV/SILV. We used two ventilators in the flow-triggered assist-control mode with no synchronizing device. Synchronization was achieved by the patient who triggered both ventilators simultaneously. Nishimura et al[27] evaluated the performance of the Servo 300 ventilator using a pediatric lung model with a 4-mm-ID endotracheal tube. The maximum pressure drop needed to trigger the ventilator was 0.42 cm H_2O [0.04 kPa] and the delay between initiation of inspiration and onset of flow was 49 ms. The minimal delay time and triggering pressure enabled our patient to initiate synchronous breaths with low inspiratory effort. Furthermore, because respiratory rate and I:E were not fixed, the patient was able to adopt the expiratory time dictated by the time constant of the individual lung.

Although Hillman et al[20] showed that there was no need for synchronization, and that asynchronous ILV had no disadvantages compared to SILV, they stated that during asynchronous ILV patients had a greater tendency to resist ventilation. In our patient, ease of synchronization may have been the primary reason that ILV was tolerated without the heavy sedation found necessary by other authors.[1,20] We were able to combine the easier handling and greater flexibility in adjusting the ventilators of asynchronous ILV with the more physiologic breathing pattern of patient-triggered SILV.

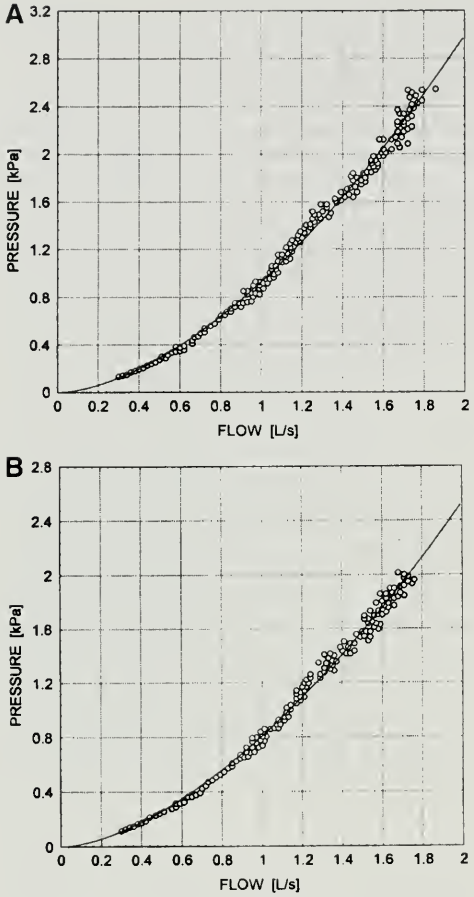

Fig. 5. Pressure-flow relationships of a 39-Fr 85-mm double-lumen tracheostomy tube. The continuous line was derived by regression analysis and fitting measurements to a power function of the form $P = a\dot{V}^b$, where P is the pressure drop across the tube, $\dot{V}$ is flow, a is the pressure at $\dot{V}$ = 1 L/s, and b is a dimensionless number that describes the shape of the curve. (A) For the bronchial lumen, a = 0.81, b = 1.64, and r^2 = 0.996. (B) For the tracheal lumen, a = 0.93, b = 1.68, and r^2 = 0.996.

Conclusion

This case demonstrates the provision of SILV using flow-triggered, pressure-regulated, assist-control mode ventilation. In our patient, ILV led to an impressive improvement in oxygenation, greatly facilitated mobilization of secretions, and made it possible to switch back to conventional ventilation after only 2 days. Although our patient's response to ILV was dramatic and even remarkable, it provides only anecdotal information, and well-controlled research is needed to document its true worth. Although ILV is more labor-intensive and therefore more costly, we believe that the increased costs will be offset by a reduced length of stay in the ICU for patients such as ours.

ACKNOWLEDGMENTS

We are indebted to Robert M Kacmarek PhD RRT for valuable comments on this manuscript.

PRODUCT SOURCES

Ventilators:

Servo 300, Siemens Medical Systems Inc, Danvers MA
Servo 900C, Siemens Medical Systems Inc, Danvers MA

Tracheostomy Tubes:

Tracheopart double-lumen 39-Fr (85-mm), Rusch Inc, Duluth GA

Pharmacologic Agents:

Ofloxacin, Tarivid, Hoechst AG, Frankfurt, Germany
Midazolam, Roche Laboratories, Nutley NJ
Sufentanil, Janssen Pharmaceutica, Titusville NJ
Penicillin G, Wyeth Ayerst Laboratories, Philadelphia PA
Imipenem/cilastatin (Zienam), Merck-Sharp & Dome, Munich, Germany
Amphotericin B, Apothecon, Bristol-Myers Squibb Co, Princeton NJ

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The Use of Exogenous Surfactant in Three Pediatric Patients following Gastric Aspiration

Neal J Thomas MD, Sally A Webb MD, Jean Smith RRT, Fred W Tecklenburg MD, and David M Habib MD

Mortality due to ARDS of all etiologies is at least 50%. However, mortality due to ARDS induced by pulmonary aspiration of gastric contents is reportedly greater than 90%. We report the cases of 3 children who aspirated gastric contents and who were subsequently treated with intratracheal surfactant in the hope of reversing the pulmonary dysfunction—hypoxemia and low compliance—associated with their ARDS. All 3 patients demonstrated improvement in oxygenation and compliance after the first dose of surfactant and 2 survived without apparent pulmonary morbidity; 1 patient did not survive but did show transient improvement. Well-controlled clinical trials are needed to expose the therapeutic benefit of surfactant therapy in ARDS from aspiration of gastric contents. [Respir Care 1994;39(9):912-916.]

Introduction

Mortality from the adult respiratory distress syndrome (ARDS) remains unacceptably high at 50% or more.[1-3] Although a variety of different insults may lead to ARDS, a common final result is alveolar-epithelial and pulmonary-microvascular-endothelial injury.[1] Ample evidence from human studies and lung injury models of ARDS suggest that abnormal surfactant function directly contributes to the pathophysiology of the lung dysfunction seen in patients with ARDS.[4-7] This pulmonary dysfunction manifests as refractory hypoxemia and decreased pulmonary compliance.[2]

In ARDS induced by aspiration, there is little clinical information regarding the efficacy of surfactant replacement. Preliminary data suggest that replacement with artificial surfactant improves gas exchange[8] and lung recoil.[9] We report three cases of ARDS resulting from gastric acid aspiration in which artificial surfactant was used and may have contributed to the subsequent improvement in oxygenation and pulmonary compliance.

Case 1

PT was a 21-day-old boy who was well until approximately 1 hour prior to admission when his parents noticed his choking during a postprandial emesis. Mouth-to-mouth resuscitation was begun by the parents and on emergency room arrival, the patient was cyanotic with poor respiratory effort and was intubated because of persistent apnea.

Upon arrival in our Pediatric Intensive Care Unit, the patient's alveolar-arterial oxygen gradient [$P_{(A-a)O_2}$] was severely widened at 550 torr (Fig. 1), pulmonary compliance was poor at 0.17 mL · cm H_2O^{-1} · kg^{-1} (Fig. 2), and a chest radiograph showed diffuse consolidation of both lungs, which quickly progressed to diffuse radiographic granularity throughout both lungs—a finding consistent with ARDS (Fig. 3).

The patient's respiratory status continued to decline with hypoxemia refractory to peak inspiratory pressures ranging from 50-60 cm H_2O [4.9-5.88 kPa] and 100% oxygen. On Hospital Day 3, the patient was given three 100-mg/kg doses of exogenous surfactant (Survanta, Ross Laboratories, Columbus OH) intratracheally at approximately 8-hour intervals. Marked clinical (Figs. 1 & 2) and radiographic (Fig. 4) improvement was observed after the first dose.

Dr Thomas is a resident physician, Dr Webb is a pediatric intensivist, and Dr Tecklenburg is Director of Pediatric Critical Care, Ms Smith is a clinical supervisor and Dr Habib is Medical Director, Department of Pediatric Respiratory Therapy—Medical University of South Carolina Children's Hospital, Charleston, South Carolina.

None of the authors has a financial interest in any of the products mentioned in this paper.

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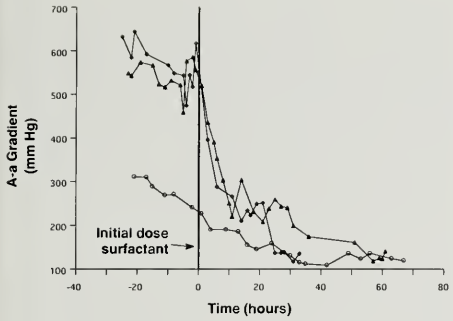

Fig. 1. A-a oxygen gradient narrows in three patients following surfactant administration. Note the improvement after the initial dose.

$$\text{A-a gradient} = P_{AO_2} - P_{aO_2},$$
$$\text{where } P_{AO2} = [P_B - P_{H_2O}] \times [F_{IO_2} - P_{aCO_2}] \times 1.25.$$

Case 1 = ○ , Case 2 = ▲ , and Case 3 = ◆ .

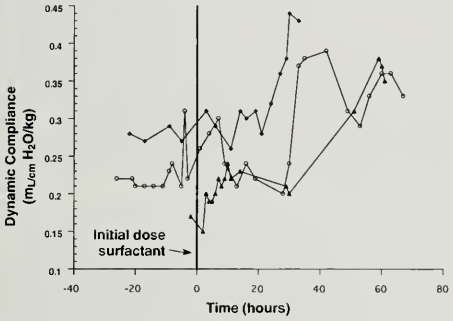

Fig. 2. Dynamic compliance (C_{dyn}, indexed to body weight) changes in three patients following surfactant administration. Although a trend toward improvement can be seen, it may not be related to surfactant administration.

$$C_{dyn} = \frac{V_T}{PIP - PEEP},$$

where V_T= exhaled tidal volume in M_L, PIP = peak inspiratory pressure in cm H_2O, and PEEP = positive end-expiratory pressure in cm H_2O.

Case 1= ○ , Case 2 = ▲, and Case 3 = ◆ .

On Hospital Day 11, he was extubated and subsequently discharged on Day 17. He required no supplemental oxygen or medications.

Case 2

RD was a 22-month-old boy with a diagnosis of ventricular septal defect and double-chamber right ventricle who was admitted for definitive surgical repair. On admission, the patient was in stable condition with no respiratory difficulty. On Hospital Day 2, he underwent surgical correction of his cardiac defects without incident. He was extubated on Postoperative Day (POD) 1 and transferred to the pediatric cardiothoracic floor.

On POD 4, the patient experienced acute respiratory decompensation, was found to have gastric contents in his oropharynx, and was intubated immediately. The postintubation chest radiograph revealed extensive bilateral alveolar opacities consistent with a diagnosis of ARDS. He de-

Fig. 3. Chest radiograph of Case 1, 5 hours prior to treatment with surfactant when peak inspiratory pressure was 50 cm H_2O [4.9 kPa] and positive end-expiratory pressure was 6 cm H_2O [0.56 kPa].

Fig. 4. Chest radiograph of Case 1, 6 hours after initial dose of surfactant when peak inspiratory pressure was 47 cm H_2O [4.6 kPa] and positive end-expiratory pressure was 6 cm H_2O [0.56 kPa].

veloped refractory hypoxemia despite the use of high levels of positive end-expiratory pressure (PEEP) and pressure-control inverse-ratio ventilation. On POD 5 he received three 100-mg/kg doses of exogenous surfactant (Survanta) intratracheally at about 8-hour intervals. This patient experienced improvement in oxygenation (Fig. 1) and dynamic compliance (Fig. 2), weaning of his ventilatory support, and partial clearing of the diffuse pulmonary disease previously noted on chest radiograph.

However, the patient's respiratory status declined on POD 9 when hypoxemia developed despite increasing ventilatory support. Serial chest radiographs were consistent with the fibroproliferative phase of ARDS. He received two additional doses of surfactant on POD 12, with no change in oxygenation or compliance.

The patient continued to do poorly with worsening pulmonary function, ventilation, and oxygenation complicated further by multiple pneumothoraxes. He decompensated slowly and expired on POD 49.

Case 3

RR was a previously healthy 16-month-old boy who was brought to our PICU from an outlying hospital after ingesting 22 quinidine slow-release tablets of 300 mg each (total of 6,600 mg or 508 mg/kg). Prior to his arrival in our emergency room, the patient had been intubated and resuscitated because of hypotension and a wide QRS complex on electrocardiogram. The patient was also given charcoal by nasogastric tube.

Upon arrival in our intensive care unit, the patient was in full cardiopulmonary arrest, requiring resuscitation. During the resuscitation, his endotracheal tube became dislodged, and he subsequently vomited, resulting in his aspirating charcoal and other gastric contents.

The patient's condition remained extremely labile with severe dysrhythmias refractory to overdrive pacing and hypotension that did not respond to high-dose catecholamines. The patient was placed on extracorporeal membrane oxygenation (ECMO) and had increasing pulmonary edema with only marginally acceptable P_{aO_2} and oxyhemoglobin saturation despite 50-60% extracorporeal bypass.

By Hospital Day 6, hemodynamic stability was achieved with this patient. Attempts to wean from ECMO were unsuccessful because the patient's oxygenation remained poor (P_{aO_2} of 45 torr on F_{IO_2} 0.5) on PEEP 14 cm H_2O [1.37 kPa]. Serial chest radiographs revealed bilateral parenchymal consolidation, and values for dynamic compliance remained low (Fig. 2). On Hospital Day 9, three 100-mg/kg doses of surfactant (Survanta) were administered intratracheally at approximately 12-hour intervals. The patient experienced marked improvement in oxygenation and ventilation (Fig. 1). On Hospital Day 13, ECMO was discontinued and the patient was decannulated. On Hospital Day 21, he was extubated and given 30% oxygen. On Hospital Day 29, he was discharged home without the need for supplemental oxygen.

Discussion

A variety of different insults may lead to ARDS but a common etiology in pediatrics is pulmonary aspiration of gastric contents. Mortality for this subset of ARDS patients is reportedly greater than 90%.[8] Aspiration of a large volume of acidic gastric contents is characterized by deterioration of gas exchange and requirement of assisted ventilation with high F_{IO_2}, high peak inspiratory pressure (PIP), and PEEP.[10] Hydrochloric acid (HCl) causes immediate damage to the alveolar-capillary membrane. These events lead to the influx of protein-rich edema fluid into the alveoli, inhibiting surfactant function.[11] As a result, alveolar surface tension increases, causing atelectasis, ventilation-perfusion abnormalities, decreased lung volumes and compliance, and hypoxemia.

In ARDS, secondary alterations of surfactant include changes in surfactant pool size, phospholipid composition, and altered ratio of large to small surfactant aggregates.[12] Although the lung injury in ARDS is complex, these secondary alterations of the endogenous surfactant system contribute to the pathophysiology of lung dysfunction seen in aspiration syndromes. Also, the functional activities of the recovered surfactant have correlated closely with the degree of respiratory failure as evidenced by measurements such as gas exchange, surface tension, and dynamic compliance.[3]

Studies using animal models of ARDS[13-16] (created using lung lavage, hyperoxia, and N-nitroso-N-methylurethane) have demonstrated that exogenous surfactant replacement therapy improves gas exchange and lung mechanics, attenuates alveolar permeability, and reduces mortality. Overall, these studies have shown that early surfactant administration may prevent the progression to ARDS.

The positive results of surfactant therapy are even more pronounced in studies using animal models of pulmonary acid aspiration. Eijking and colleagues[8] showed in rats that tracheal instillation of 0.1N HCl caused significant deterioration of oxygenation that was best prevented by administration of surfactant within 10 minutes of acid aspiration. However, after 90 minutes of deteriorating gas exchange, little or no beneficial effect was found following surfactant replacement. The protein concentration in bronchoalveolar lavage (BAL) fluid of animals receiving HCl was significantly elevated, consistent with an influx of protein-rich edema fluid into the alveolar space. Their results suggest that surfactant replacement therapy should be used early following aspiration of gastric contents to prevent development of respiratory failure.[8] Because it removes alveolar-inhibitory proteins, BAL may be associated with an improved response to exogenous surfactant replacement therapy.[11]

Limited clinical experience is available that attests to the success of administration of exogenous surfactant to humans with ARDS. Case reports in adults have described transient or limited improvement in gas exchange following exogenous surfactant administration in ARDS caused by aspiration, sepsis, burns, and pancreatitis.[17,18] From two prospective placebo-controlled trials, using aerosolized surfactant, investigators have observed narrowing of A-a gradient, improvement in respiratory compliance, and a trend toward decreased mortality in septic ARDS patients.[19,20] Improved oxygenation was reported in two children who received exogenous surfactant following near-drowning and pulmonary hemorrhage.[21,22]

In our experience with patients who aspirate gastric contents reported here, exogenous surfactant was well tolerated and was followed by improvement in pulmonary gas exchange. Surfactant was administered intratracheally in doses of 4 mL/kg, with volumes as high as 50 mL. Rapid improvement in oxygenation as shown by the narrower A-a gradient followed the initial dose of surfactant in two of our three patients (Fig. 1). The changes in values of these variables were less apparent for Case 3 due to lower baseline P_{aO_2} and insufflation pressures while on ECMO (Fig. 1).

In all patients, baseline values for dynamic compliance were markedly reduced (Fig. 2), varying between 0.15 and 0.30 mL · cm H_2O^{-1} · kg^{-1}. In contrast to the rapid changes detected in oxygenation, at least 3 doses of surfactant were necessary before sustained improvement in dynamic compliance was observed. Therefore, in these three patients, improvement in oxygenation appeared earlier than changes in dynamic compliance.

It is also important to note that exogenous surfactant was given to all patients at least 24 hours after aspiration. The positive response of our patients differs from reported experience in animals in whom no beneficial effects were observed if treatment was delayed beyond 90 minutes.[8]

In conclusion, there is increasing clinical and laboratory information suggesting that patients who experience severe pulmonary aspiration of gastric contents, an etiology of ARDS, may respond to surfactant-replacement therapy. To further evaluate the efficacy, timing, and dose-response of exogenous surfactant, carefully designed studies of animal models of gastric-acid aspiration and prospective human trials are necessary.

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CORRECTIONS

The names of the working group were inadvertently omitted from the Static Lung Volumes CPG published in the August issue of RESPIRATORY CARE [Respir Care 1994;39(8):830-836]. They are shown below. We regret the error.

Cardiopulmonary Diagnostics Committee:

Kevin Shrake MA RRT, Chairman, Springfield IL
Sue Blonshine BS RRT RPFT, Lansing MI
Robert A Brown BS RRT RPFT, Madison WI
Michael J Decker CRTT, Cleveland OH
Gregg L Ruppel MEd RRT, St Louis MO
Jack Wanger MBA RRT RPFT, Denver CO

In the August 1994 issue of the Journal, the reference issue for Question 22 was listed in error as December 1993. The information upon which this question is based was taken from Test Your Radiologic skill by Brooks and Mishoe in the November 1993 issue, Pages 1201-1204. We regret the error and any confusion it may have caused.

Letter on topics of current interest or commenting on material in RESPIRATORY CARE, will be considered for publication. The Editors may accept or decline a letter or edit without changing the author's views. The content of letters as published may simply reflect the author's opinion or interpretation of information—not standard practice or the Journal's recommendation. Authors of criticized material will have the opportunity to reply in print. No anonymous letters can be published. Type letters double-spaced, mark it "For Publication," and mail it to RESPIRATORY CARE Journal, 11030 Ables Lane, Dallas TX 75229-4593.

Letters

RCPs, CPR and Research on Mouth-to-Mask Devices

To further emphasize the importance of involving respiratory care practitioners in resuscitation as encouraged by Barnes and Durbin in the July issue of the Journal,[1] we would like to share our experience involving mouth-to-mask devices.

In the course of determining the oxygen concentration delivered (F_{DO_2}) at various flowrates, we observed several potentially harmful situations that could occur during mouth-to-mask resuscitation.[2] First, when the design of the device is such that the oxygen port is located above (toward the operator) from the duck-bill (or one-way valve), passive exhalation cannot occur. We theorized that oxygen flow was sufficient to keep the duck-bill valve open and the expiratory valve closed. This problem was corrected by inserting the supplemental oxygen port between the duck-bill valve and the face mask. Second, supplemental oxygen flow (whether it is 5 L/min or 15 L/min) augments the rescuer's delivered breath from 25 to 230%.[2,3] This may explain why tidal breaths delivered by mouth-to-mask techniques commonly exceed those attained by bag-valve-mask ventilation.[4,5] Finally, the F_{DO_2} during mouth-to-mask ventilation is influenced not only by the oxygen flowrate but also (and more importantly) by the size of the rescue breath and the rate of rescue breathing. Although these situations may not appear important in the adult population, we suggest that mouth-to-mask devices be used with caution in the infant and pediatric age groups. Because of the potential risk of gastric insufflation, gastric perforation, and pneumothorax, we recommend that for infants and small children the supplemental oxygen port be placed between the duck-bill and the face mask and that oxygen be added at a rate of 5 L/min or less—particularly because little increase in F_{IO_2} is achieved at higher flows.

Despite the simple design of mouth-to-mask devices, serious injury may result from what appears to be a simple, logical modification to improved F_{DO_2}. Because of this, we encourage respiratory care practitioners to thoroughly investigate all modifications before patient application—no matter how benign they may appear.

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Nosocomial Cutaneous Mucormycosis at the Site of Endotracheal Tube Taping

We report an unusual but devastating complication of endotracheal tube management related to tube taping. This case, in more detailed form, is being published in the journal *Cutis* in which the association of mucormycosis with iron overload states is emphasized.[1] We wish to highlight the important clinical aspects of this unfortunate case.

Case Report

A previously healthy 61-year-old Japanese woman presented with painless jaundice, pruritus, and weight loss due to a Klatskin tumor involving the right hepatic duct. Following a partial debulking procedure with a Roux-en-Y reanastomosis, the woman had a long and complicated postoperative course. Early complications included small bowel obstruction, right hemothorax, high-output enterocutaneous fistula, protein-calorie malnutrition, and several nosocomial infections in which a variety of organisms were cultured from her sputum, pleural fluid, gastrointestinal tract, urine, operative wound, and blood. Her treatment included multiple courses of broad spectrum intravenous antibiotics and high-dose supplemental insulin therapy to maintain her labile blood glucose levels within euglycemic limits.

Approximately 4 months after the initial procedure, her clinical status was further compromised by progressive renal insufficiency and worsening liver failure. After intubation, a right thoracotomy with decortication was performed to treat the persistent hemothorax.

On the 139th hospital day, small bullous lesions were noted on her upper lip surrounding the surgical tape securing her oral endotracheal tube. These were initially attributed to "tape burns," but over the ensuing 48 hours rapidly coalesced into a black, necrotic eschar. Punch biopsy confirmed a diagnosis of cutaneous mucormycosis. Because of the gravity of her condition, our patient was considered an unacceptable surgical candidate for the extensive debridement, and family and caregivers elected to withdraw care. The patient expired 2 days later.

Discussion

The nosocomial transmission of cutaneous mucormycosis is a rare but frequently fatal event that classically occurs in critical-

ly ill patients at the site of either a surgical wound or dermatologic trauma.[2] Our case is somewhat unusual in that the infection occurred under the surgical tape securing an oral endotracheal tube where there had been no previous dermatologic trauma. This incident is similar to the description of 6 cases of primary cutaneous mucormycosis that occurred in patients with wounds treated with contaminated Elastoplast™ dressings.[3]

Similar to other reports, the clinical course of the infection was a rapid progression from an erythematous macule to a necrotic eschar.[4] Punch biopsy was used to confirm the diagnosis because wound swab and culture results are often unrewarding.[5] Effective therapy for established primary cutaneous mucormycosis infection is extensive surgical debridement and aggressive intravenous amphotericin-B administration.[6] Some authors suggest that if the infection is discovered while it is still superficial, surgical debridement combined with topical amphotericin-B may be adequate therapy.[7] Primary cutaneous mucormycosis originating in the face and perioral region is especially morbid; a recent study found that 100% of primary infections arising on the head and trunk were fatal.[8]

We believe that our case should be of interest to respiratory care providers for three reasons. First, although primary cutaneous mucormycosis is a rare entity, it is a potentially fatal nosocomial infection and should be considered in a susceptible host. Second, the cutaneous form usually occurs at the sites of surgical wounds or tissue trauma but can also arise in mildly or nontraumatized tissue. Third, our case illustrates a little known but devastating complication of endotracheal tube management that may first be noted by practitioners of respiratory care.

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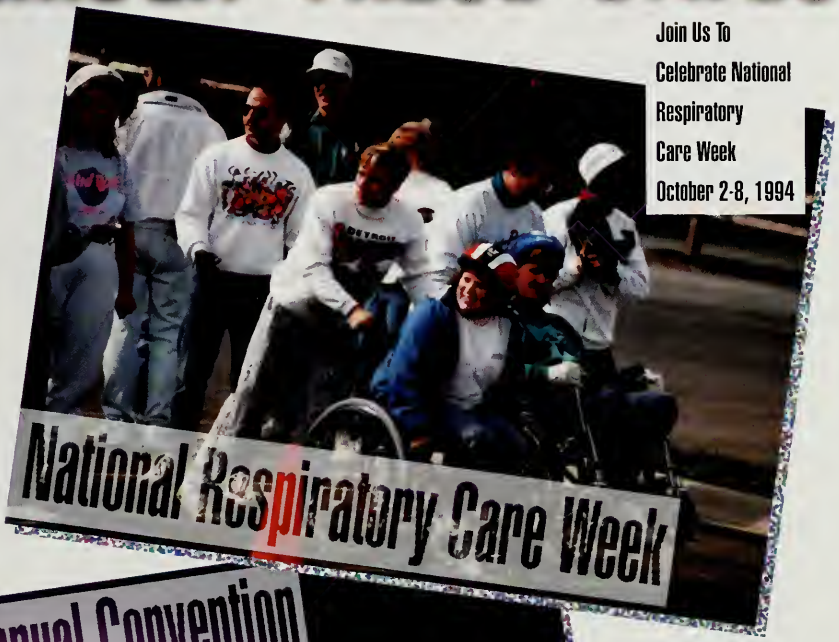

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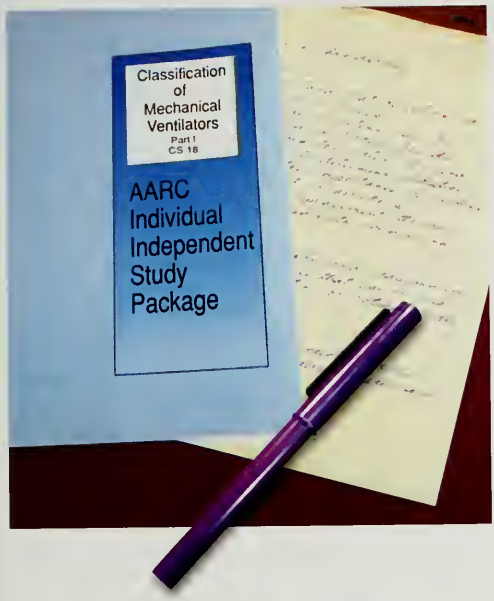

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Learn how to compute and record static compliance curves and dynamic characteristics. Also learn the procedure of obtaining pressure-volume measurements and interpret the compliance characteristics measurements.
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Describes the four routes of tracheal intubation and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.
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Covers the selection of the proper equipment necessary to perform endotracheal intubation and to ensure that they are in working order.
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Type on one side of white bond paper, 216 5 279 mm (8 in. 5 11 in.) with margins of at least 25 mm (1 in.) on all sides of the page. ***Double-space*** the entire manuscript (three lines per vertical inch). Number all pages in upper-right corners. Indent paragraphs 5 spaces. Do ***not justify***. Do ***not*** underline titles, headings, or other words. Do ***not*** type authors' names or other identification anywhere except on the title page. Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: title page, abstract, text, product-sources list, acknowledgments, reference list, each table, each appendix, list of figure captions. Use standard English. Employ the first person and active voice (eg, "We believe that pigs can fly") rather than the 'obscure person' and passive voice (eg, "It is believed that pigs can fly")—because the latter obscures the identity of the responsible party (the believer).

Headings in Text: Center ***main section headings*** on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin ***subheadings*** at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis). Do ***not underline or darken*** section headings or subheadings.

Manuscript Structure

Most kinds of papers have standard parts in a standard order, as shown hereafter. However, papers can vary individually, and not all papers will have all the parts listed here.

Research Article: Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figure Legends.

Evaluation of Device/Method/Technique: Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figure Legends.

Case Report: Title Page, Introduction, Case Summary, Discussion, References, Tables, Figure Legends.

Review Article: Title Page, Table of Contents, Introduction, Review of the Literature, State-of-the-Art Summary, Acknowledgments, References, Tables, appendices, and illustrations may be included. Other formats may be suitable.

Point of View Paper: Title Page, Text, References. Tables and illustrations may be included.

Details about Sections:

Title: Make the paper's title as specific, clear, and yet as short as you can.

Title Page: List (a) title of the paper; (b) full names of all authors, with academic and credential letters, professional titles, and institutional affiliations; (c) name, address (include building and/or room number for courier service), telephone number, and Fax number of corresponding author; (d) name and address for reprint requests; (e) sources of support such as grants, equipment, drugs, and supplies; (f) name of organization, location, and date of any meeting at which a version of the paper has been presented; (g) disclosure of financial relations of any author with commercial products or interests connected with the paper—or with competing products or interests; (h) name, title, and affiliation of statistical consultant, if any; and (i) disclaimers, if any.

Abstract: (required only for research articles and evaluations of devices/ methods/techniques). The abstract must summarize what was studied; why and how it was studied; the results, including important data and statistical significance; and conclusions drawn from the results. All information in the abstract must also appear in the paper itself. Do not cite references in the abstract. The abstract for a research article should include the following headings (in all capital letters), appropriately placed within the abstract and followed by colons: **BACKGROUND, METHODS, RESULTS, CONCLUSIONS.** The abstract for a paper evaluating a device/method/technique should include the following headings: **BACKGROUND, DESCRIPTION OF DEVICE, EVALUATION METHODS, EVALUATION RESULTS, CONCLUSIONS.** The abstract should be all one paragraph, not indented, and not longer than 250 words. Center title, typed in capital and lower case letters, over abstract.

Introduction: Briefly describe the background of the work or the paper. Cite only pertinent references, and do not review the subject extensively. Do not include data or conclusions from the work reported in your paper. In a research paper, end this section with a clear statement of the research question(s) or hypothesis(es).

Methods Section (in a research paper): Describe the selection of patients, controls, or laboratory animals. Give details about randomization. Describe methods for blinding of observations. Give numbers of observations. Report losses to observation (eg, dropouts or disqualified subjects), listing numbers of subjects or data sets lost, when lost, and why lost. *Describe methods in sufficient detail to allow other workers to replicate your work.* Give references to established methods; provide references and brief descriptions for methods that have been published but are not well known; describe new or substantially modified methods, give reasons for using them, and evaluate their limitations. Report calibration of measuring devices.

Drugs—Identify precisely all drugs and chemicals used, giving generic names, doses, and routes of administration. If desired, brand names may be given in parentheses after generic names.

Commercial Products—Identify any commercial product (including model number if applicable) the first time it is mentioned, giving the manufacturer's name, city, and state or country—in parentheses in the text. If four or more products are mentioned, do not list any manufacturers in the text; instead, list them on a Product Sources page at the end of the text before the References. Provide model numbers when available and manufacturer's suggested price if the study has cost implications.

Ethics—When reporting experiments on human subjects, indicate that procedures were in accordance with the ethical standards of the institution's committee on human experimentation. State that informed consent was obtained after the nature of the procedure(s) had been explained. Do not use patient's names, initials, or hospital numbers in text or illustrations. When reporting experiments on animals, indicate that the institution's or any national guide or national law on the care and use of laboratory animals was followed.

Statistics—In the last paragraph of the Methods section, identify the statistical tests used in analyzing the data, and give the prospectively determined level of significance. Cite references to support choices of tests. (Cite textbooks or published articles, not handbooks of commercial software.) Identify any general-use or commercial computer programs used, naming manufacturers and their locations.

Results Section: Present results in logical sequence in the text. Tables and illustrations may also present data. Do not repeat in the text all the data in the tables or illustrations; emphasize or summarize only important observations and trends. Be sure to report all the results; do not save some of them for the Discussion section. Do not discuss the findings in the Results section. Exact p values are preferred in all cases but are *essential* when values are not statistically significant. Do not report original results merely as nonsignificant or NS.

Discussion Section: It may be useful to restate the research question(s), but do not repeat in detail the data or other material given in the Introduction, Methods, or Results sections. Emphasize the new and important aspects of the study and the conclusions that follow from them. Present the implications and limitations of the findings—including implications for future research. Relate the findings to other relevant published work. Link the conclusions with the goals of your work, but avoid unqualified statements and conclusions not completely supported by your data. Avoid claiming priority and alluding to work that has not been completed. State new hypotheses when warranted, but clearly label them as such. Recommendations, when appropriate, may be included. Provide a clear 'take-away' message for readers—either at the end of the Discussion section or in a separate **Conclusions** section.

Product Sources Page: When four or more commercial products, including statistical software, are mentioned in the paper, list manufacturers' names, cities, and states or countries on a Product Sources page after the text. For each kind of product, list the generic term, brand name and model number, manufacturer's name, city, and state or country. Manufacturer's suggested price should be included when the study or evaluation has cost implications. For example:

Manual Resuscitators:
BagEasy, Respironics Inc, Murrysville PA, $20.50
Code Blue, Vital Signs Inc, Totowa NJ, $19.85

Ventilators:
7200, Puritan-Bennett Corp, Overland Park KS
Bear Cub, Bear Medical Systems, Riverside CA

Acknowledgments Page: On this page you may recognize the services of persons who made ancillary contributions to the work or the manuscript. Such services might be advice about methodology; data collection; statistical advice or analysis; equipment selection or operation; cooperation as caregiver, patient, or subject; manuscript preparation; in-house review; and other services. Each acknowledgment must specify the service rendered. Named persons must provide written agreement (accompanying submitted manuscript) to be so recognized.

References

Use of References: References are used to support statements of fact, to indicate sources of information, or to guide readers to further information. Be careful to make clear in the text the reason for a specific citation (ie, do not imply support of a statement of fact by citing a reference that simply addresses the issue). Cite only sources that have actually been consulted and evaluated by the authors. Cite only ***published or accepted*** material. Cite original articles in preference to textbooks, review articles, abstracts, editorials, or letters. Avoid citing abstracts because some published abstracts have never been subjected to peer review and may not support your contention. Make every effort to determine whether an abstract has been subsequently published as a full-length paper. Avoid citing non-English language sources. When citing from a book, specify the page numbers unless you are citing the entire book. If you cite a paper that has been accepted but not yet published ("in press"), provide a copy of the paper to the Editor when you submit your manuscript.

Do not cite unpublished observations as references. Instead, identify written (not oral) communications in parentheses in the text, giving the writer's name and location and the date of the communication. Information from manuscripts submitted but not yet accepted should be cited in the text (in parentheses) as "unpublished observations."

Citing References in the Text: The first reference you cite is Reference 1, the next is Reference 2, etc. After the first citation of a reference, use its original number if you cite it again later in the paper. Cite references by superscript, full-size, arabic numerals. **Do not enclose in parentheses.** If a citation numeral is located at the end of a phrase or sentence, place the numeral after (outside) the comma, semicolon, or period—not before (inside) it. Avoid citing references at the end of a phrase or sentence if they pertain only to internal parts of the phrase or sentence; instead, cite them at the pertinent places within the phrase or sentence (eg, Hess et al[13] discovered that . . .").

Listing References: Starting on a new page after the text, list the references in numerical order. Do not employ "op cit" or "ibid." Type references double-spaced, using the styles of the examples given hereafter. List all authors (do not use "et al"). In titles of articles and books, capitalize only first words and proper names. Abbreviate journal names as in Index Medicus. Spell out in full the names of less well known or nonindexed journals and periodicals If the cited item is an abstract, editorial, or letter, identify it as such in parentheses following the item's title. Provide both first and last complete page numbers. Do not leave spaces between dates and volume and page numbers. Obtain authors' names, article and book titles, dates, and volume and page numbers from the *original* cited articles and books, not from other articles' reference lists, which often are inaccurate. Examples of correct reference listings follow (these are single-spaced here but must be *double-spaced* in a manuscript).

Article in a journal carrying pagination throughout volume:

1. Shepherd KE, Johnson DE. Bronchodilator testing: an analysis of paradoxical responses. Respir Care 1988; 33:667-671.

Article in publication that numbers every issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139s-143s.

Abstract in journal: (Abstracts are not strong references. Abstracts more than 3 years old should not be cited. When cited, abstracts should be identified as such.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35:1087-1088.

Editorial in journal:

6. Rochester DF. Does respiratory muscle rest relieve fatigue or incipient fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Editorial with no author given:

7. High frequency ventilation (editorial). Lancet 1991; 1:706-708.

Letter in journal:

8. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (Specific pages should be cited whenever possible.)

10. Nunn JF. Applied respiratory physiology. New York: Appleton-Century Crofts, 1969.

Note: To specify pages in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1969:85-95 (series of contiguous pages), 1969:85,95 (separated pages).

Corporate author book: (Specific pages should be cited whenever possible.)

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with editor(s): (Specific pages should be cited whenever possible.)

12. Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in book: (Specific pages should be cited whenever possible.)

13. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Phildelphia: JB Lippincott, 1977:171-223.

Newspaper article:

14. Rensberger B, Specter B. CFCs may be destroyed by natural process. The Washington Post 1988 Aug 7:Sect A:2(Col 5).

Dictionary or similar reference:

15. Pneumohemopericardium. Dorland's illustrated medical dictionary, 26th ed. Philadelphia: WB Saunders, 1981: 1038.

Tables: Use tables to display information, compare data, or show trends. Start each table on a separate page. Do not construct a table with fewer than two lines (rows) or columns of data (instead, put the data in the text). Avoid more than 8 columns across. Number tables as Table 1, Table 2, etc, consecutively in the order of their first mention in the text. Place the number and a **descriptive** title above the table (not on a separate page). Give each column a brief heading. Place explanatory matter in footnotes, not in the title or column headings. Explain in footnotes all nonstandard abbreviations and symbols used in the table. To key footnotes to the table body, use conventional designations (aster-

isk, dagger, double dagger, etc) in consistent order, placing them superscript in the table body.

Double-space all elements of tables, including titles, column headings, data, and footnotes. Continue a deep table on following pages. Do not use horizontal or vertical rules. Do not submit tables as photographs, or reduced in size, or on oversize paper. Use the same typeface as in the text. Supply the name and version of any table-building computer program used.

Appendices: Mathematical calculations, documents, and other matter that would clutter the main article can be displayed in appendices. Number them as Appendix 1, Appendix 2, etc, and refer to them in the text. Give each appendix a descriptive title and type it double-spaced throughout.

Illustrations: Graphs, line drawings, photographs, and radiographs are called figures. Use only illustrations that clarify and augment the text. Number them consecutively as Fig. 1, Fig. 2, etc, according to the order in which they are first mentioned in the text. Figures for publication must be of professional quality, but rough sketches may accompany the submitted manuscript, with final figures to be prepared after review. Figures need not be photographic reproductions. Clear, clean laser-printer-generated figures are acceptable (121-144 dpi). However, the data from which the original graphs are generated should be available to the editor upon request. Remember that originals that are roughly 7 5 9 inches will be reduced to less than 50% (3 5 4 in) and originals with a horizontal dimension of 9 in will be reduced to less than 33%. Photographs must be glossy 5 5 7 to 8 5 10-inch black and white prints, unless color is essential. (If color is essential, consult the Editor to learn whether negatives, transparencies, or prints are required.) In reports of animal experiments, use schematic drawings, not photographs. A letter of consent must accompany any photograph in which a possibility of identification of a person exists; masking the eyes is not sufficient. Lettering and numerals must be neat, uniform in size and style, and large enough to remain legible when downsized for publication. Do not place titles and detailed explanations on figures; put such information in the figure captions. Identify each figure on the back with a stick-on label showing figure number, an arrow indicating the top, and an abbreviated manuscript title. ***Omit author's name.*** Cover label with clear tape so ink will not smudge other prints. Do not use staples or paper clips, and do not write heavily on the backs of prints.

Radiographs: If possible, submit radiographs as full-size copies of films, not as prints. Prints may be acceptable, but full-size films are preferable in order to display better detail in published figures. Be sure all figures are cited in the text. If any figure has been published before, include copyright-holder's written permission to use it.

Figure Captions: Its caption should, to the extent possible, make a figure understandable without referring the reader to the text. Type figure captions double-spaced, on a separate page, as Fig. 1, Fig. 2, etc. When symbols, arrows, numbers, or letters are used to identify parts of a figure, identify and explain each part clearly in the caption. In photomicrographs, explain the internal scale and method of staining. If a figure has been published before, acknowledge the original source in its caption (permission must be obtained prior to use, of course).

Units of Measurement: Give measurements of length, height, weight, and volume in metric units appropriately abbreviated. Give temperatures in degrees Celsius. Give blood pressures in millimeters of mercury (mm Hg). Report hematologic and clinical-chemistry measurements in conventional metric system and in SI units (International System of Units). Show gas pressures (including blood gas tensions) in torr. List SI equivalent values, when possible, in brackets following non-SI values—for example, "PEEP, 10 cm H2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988) and 1989;34:145 (Feb 1989).

Arithmetic: Carefully double-check all arithmetic before submitting the paper. Accuracy is the author's responsibility; errors are common!

Abbreviations and Symbols: Use standard abbreviations and symbols. Avoid creating new abbreviations. Avoid all abbreviations in the title and unusual abbreviations in the abstract. Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Standard units of measurement can be abbreviated without explanation (eg, 10 L/min, 15 torr, 2.3 kPa). If you employ a great many abbreviations and symbols, provide a double-spaced list of them, with their definitions, in alphabetical order.

Please use the following forms: cm H2O (not cmH2O), f (not bpm), L (not l), L/min (not LPM, l/min, or lpm), mL (not ml), mm Hg (not mmHg), pH (not Ph or PH), $p > 0.001$ (not $p>0.001$), s (not sec), SpO2 (pulse oximetry saturation).

Computer Diskettes: A manuscript may be submitted on a Macintosh or IBM-compatible diskette. **Macintosh documents on 3.5 in. diskettes written in Microsoft Word versions 4.0 and 5.0 are preferred. However, we can convert most documents from software (including PS-DOS format) to a Macintosh-Microsoft Word document.**

Label each diskette with date; author's name; name of word-processing program and version used to prepare documents; and filename(s). If not enough space is available, list contents on disk jacket or an attached note. Do not write on a diskette except with a felt-tipped pen.

Tables and figures must be in their own separate files, with software identified.

Together with diskette, supply three hard copies of the manuscript. Do not paperclip a diskette to its hard copy.

Proofreading and In-House Review: Have all authors proofread the manuscript for content accuracy and language. Consider having the manuscript reviewed in-house by colleagues before submitting it.

Submitting the Manuscript

Use the checklist below to make sure the manuscript is ready for mailing. Mail three copies of the manuscript and figures to RESPIRATORY CARE, **11030 Ables Lane, Dallas TX 75229-4593.** Do not Fax manuscripts. Protect figures with cardboard to prevent bending. A computer diskette submission must be accompanied by the requisite three hard copies. Keep a copy of the manuscript and figures in your files in case of loss. You will be sent an acknowledgment that your manuscript has been received.

Cover Letter: The manuscript must be accompanied by a covering letter signed by all the authors. The letter must specify the intended publication category and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, proofread the accompanying manuscript, and approved its submission for publication."

Permissions: The manuscript must be accompanied by copies of permissions to reproduce published material (figures or tables); to use illustrations of, or report sensitive personal information about, identifiable persons; or to name persons in the Acknowledgments section.

Author's Checklist:

1. Does paper fit a listed publication category?
2. Does the cover letter meet specifications?
3. Is the title page complete?
4. **Is double-spacing used throughout entire manuscript?**
5. Are all pages numbered in upper-right corners?
6. Are paragraphs indented 5 spaces?
7. Are all references, figures, and tables cited in the text?
8. Are references typed in requested style?
9. Have SI values been provided?
10. Has all arithmetic been checked?
11. Have generic names of drugs been provided?
12. Have necessary written permissions been provided?
13. Have authors' names been omitted from text and figure labels?
14. Have copies of "in press" references been provided?
15. Has manuscript been proofread by all authors?

Lambda Beta Scholarships

The Lambda Beta Society, National Honor Society for the Profession of Respiratory Care, is awarding four $500 scholarships. An applicant should be a current student in or recent graduate of a school with an established Lambda Beta chapter. Criteria for selection are (1) evidence of scholarship (most recent transcript); (2) two letters of recommendation; (3) a sample of the applicant's written work (research paper *or* essay relative to respiratory care *or* an essay entitled What It Means To Be a Respiratory Care Professional). Deadline for submission is September 15, 1994. Scholarship applications are available from the school's chapter chairman.

Establishing a Lambda Beta Chapter

For information on establishing a chapter of the Lambda Beta Society, National Honor Society for the Profession of Respiratory Care, in your school, write, call, or fax the National Office of Lambda Beta Society, 1701 W Euless Blvd, Suite 300, Euless TX 76040 (pho 817-283-2835; fax 817-354-8519).

1994 Publication Awards

See page 677 of the June 1994 issue of RESPIRATORY CARE for information on the American Respiratory Care Foundation Publication Awards for 1994.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1994 Examination and Fee Schedule

	Examination Date	Application Deadline
CRTT:	November 12	September 1
RRT:	December 3	August 1
RPFT:	December 3	September 1

Fee Schedule	
Entry Level CRTT—new applicant:	$ 90.00
Entry level CRTT—reapplicant:	$ 60.00
RRT Written and Clinical Simulation—new applicant:	$190.00
Written Registry Only—new applicant:	$ 90.00
Written Registry Only—reapplicant:	$ 60.00
Clinical Simulation Only—new & reapplicant	$100.00
Entry Level CPFT—new applicant:	$100.00
Entry level CPFT—reapplicant:	$ 80.00
Advanced RPFT—new applicant:	$150.00
Advanced RPFT—reapplicant:	$130.00

	Active	Inactive
CRTT Recredentialing:	$25.00	$ 60.00
RRT Recredentialing:		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
P/P Specialty Credentialing:	$25.00	$130.00
Membership Renewal:		
CRTT/RRT/CPFT/RPFT		$ 12.00

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News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to Respiratory Care Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229-4593.

EDUCATIONAL PROGRAM. As part of an ongoing public-education effort, UVEX SAFETY LLC, a supplier of NIOSH-certified high-efficiency respirators, has issued a brochure on the TB epidemic and preventive measures and an instructional video on the Better Breathing HEPA-Tech 3010 disposable respirator. The brochure entitled, "TB and Your Safety: What You Should Know about the Current TB Epidemic," details the resurgence of TB as a major health threat, examines who is at risk, and explains protective measures those at risk must and should take to prevent transmission. The video, "HEPA-Tech 3010 Healthcare," provides step-by-step instruction on the fit-test procedure. Both the brochure and the video present information on the use of NIOSH-certified HEPA respirators used to protect those who are at risk for exposure to TB. To receive a free copy of either the brochure or the video, write to UVEX SAFETY LLC, Dept RC, 10 Thurber Blvd, Smithfield RI 02917, or call (800) 343-3411. Please mention RESPIRATORY CARE when you call.

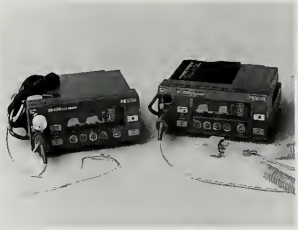
RHINOLARYNGOSCOPY PROCEDURE GUIDE. Welch Allyn Incorporated introduces a new procedure guide entitled, "The Primary Guide to Rhinolaryngoscopy." The 20-page, full-color guide discusses a variety of practitioner issues and illustrates many commonly viewed pathologies. According to the publisher, the Guide has been designed to assist practitioners by providing an overview of the in-office examination of a patient's nasal cavity, pharynx, and larynx. Welch Allyn also announces the reprinting of their catalog entitled, "Laryngoscope Collection." This catalog includes a description and glossy photo of the company's complete line of laryngoscope blades including MacIntosh, E-MacIntosh, Miller, Flagg, Guedel, Wisconsin; expendable laryngoscopes; and the complete line of fiberoptic and standard handles. For information on either of these products, call (800) 535-6663 or any Welch Allyn representative. Don't forget to mention RESPIRATORY CARE when you call.

1994 PDR GUIDE TO DRUG INTERACTIONS, SIDE EFFECTS, INDICATIONS. According to the publisher, this volume is the most comprehensive and reliable publication of its kind and is cross-referenced to the 1994 PDR. It offers up-to-date listings in three vital areas—Interactions: For a quick check of potential complications, this easy-to-access section lists drug combinations—with all brands and generic categories that could be involved. It even covers sometimes hard-to-pin-down food interactions. Side Effects: When a situation indicates a possible adverse drug reaction, simply look up the specific sign, symptom, or abnormality. You'll see whether any prescribed medicines may be the source of the problem with incidence (where supplied) to help pinpoint the most likely cause. Indications: Here are descriptions of the clinical situations you may face and a list of all drugs indicated. It helps identify the alternatives when a prescribing conflict occurs and classifies antibiotics by pathogen and system. For ordering information, write to Bill Gaffney, Dept RC, Medical Economics Data, 5 Paragon Drive, Montvale NJ 07645-1742.

TB ISOLATION ROOM MONITORS. BioSafety Systems introduces two new products of the type recommended by the Centers for Disease Control and Prevention for periodic performance checks of negative pressure isolation rooms. The Smoke Tube Kit, originally designed for respirator fit testing, contains all the necessary components to check the direction of airflow through the doorway or inside the isolation room. The Velometer, with 5% accuracy, is the ideal inexpensive anemometer for periodic air velocity measurements to confirm that the minimum 100 FPM velocity is being achieved. For more information, call Jennifer Pietrzak-Wahl at (619) 452-3500. Please mention RESPIRATORY CARE when you call.

ATTACHABLE OXIMETER PRINTER. Nonin Medical Incorporated is pleased to announce the release of the Model 8500P attachable printer for real-time documentation of spot-check or short-term-continuous pulse-oximetry data from all 8500 and 8500M hand-held oximeters. The printer slides on and off the oximeter and has a direct connection to the sensor port. Print format is data log with seven programmable options to include lowest saturation within a defined segment of time. For each measurement, actual time is indicated. Also available is an optional carrying case to hold the printer, oximeter, sensor, extra paper, and accessories. Call Theresa Neddermeyer, (612) 553-9968 and mention RESPIRATORY CARE when you call.

MINIATURE AIR-QUALITY MONITOR. VERITEQ Instruments Incorporated introduces the Spectrum 2000 data logger that can be used like a chart recorder to monitor ambient temperature and relative humidity. Such data may be used to ensure patient comfort and energy efficiency in health-care facilities. This new palm-sized monitor has no moving parts, can hold up to a year's worth of ambient temperature and humidity data for playback and graphing on an IBM or compatible computer, is self-powered and runs continuously for up to 10 years without battery replacement. For information contact Rick Schellenberg at (800) 683-8374. Please mention RESPIRATORY CARE when you call.



CO_2 MONITOR. Pryon Corporation introduces an easy-to-use, portable, and flexible CO_2 monitor—the SC-300. According to the manufacturer, the sensor reaches full operating specifications in seconds, features continuous self-calibration, and withstands excessive patient secretions and moisture without compromising accuracy. In addition to providing CO_2 measurements on intubated adults and children, the SC-300 works with nonintubated patients using the side-stream configuration and a nasal cannula. For more information, write to Pryon Corporation, Dept RC, N93 W14575 Whittaker Way, Menomonee Falls WI 53051, or call (800) PRYONCO. Please mention RESPIRATORY CARE when you call.

HEARTBEAT COUNTER. VacuMed introduces the CATEYE Heartbeat counter. It uses a unique earlobe sensor that, according to the manufacturer, is comfortable to wear and provides extremely accurate pulse readings without the need for chest belts or fingertip sensors. It can be used for immediate heart rate measurements and as a training tool for personal exercise sessions. While you exercise, the monitor measures, stores, and displays data, including the current and average pulse rate, calorie expenditure, and workout time. The clip-on sensor has two memory functions that record key heart rate data during the workout for later analysis and has high and low alarms. For information, write to VacuMed, Dept RC, 4483 McGrath Street, # 102, Ventura CA 93003.

WHOLE-BLOOD ANALYZER. Nova Biomedical introduces the Stat Profile Plus 10 analyzer to provide immediate results of a comprehensive whole-blood emergency department profile. The tests measured include blood gases, hematocrit, electrolytes, glucose, lactate, and BUN. The manufacturer believes that by consolidating the most frequently ordered emergency and critical care tests in one step, this analyzer reduces blood drawing needs, improves test turn-around time, and lowers overall testing costs. For more information, contact Lloyd Adams, Nova Biomedical, Dept RC, 200 Prospect Street, Waltham MA 02254-9141, or call (617) 904-0800. Please mention RESPIRATORY CARE when you call.

Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

AARC & AFFILIATES

September 13 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the fifth of a 6-part videoconference series: "Professor's Rounds in Respiratory Care: Hospital Operational Restructuring and Respiratory Care" (1 CRCE credit). For information, call (214) 830-0061.

September 14-16 in Ocean City, Maryland. The Maryland/District of Columbia Society of Respiratory Care presents the 13th Annual Conference by the Sea, at the Carousel Hotel. The symposium highlights reengineering, benchmarking, and therapist-driven protocols. Contact Elgloria Harrison RRT at (703) 497-2500, or Sharyon Brown CRTT at (202) 884-3125.

September 15-16 in Bethel, Maine. The MSRC presents its annual fall seminar at the Sunday River Resort. Topics include NIH asthma treatment/management guidelines, the RCP's future role in health care, pediatric BiPAP, case reviews, and health care promotion. A barbecue, dance, vendor reception, and golf tournament are planned. Participants can receive up to 10 CRCE credit hours. Contact Garry Michaud RRT, Respiratory Care Department, Penobscot Bay Medical Center, Glen Cove, Rockland ME 04841. (207) 596-8472.

September 15-16 in Boise, Idaho. The ISRC presents its annual convention at the Owyhee Plaza Hotel. Lectures, exhibits, sputum bowl competition, a poster contest, and a dance are planned. Contact Leslie Ann Sams Mulder RRT, or Connie Leavitt RRT, Respiratory Care Department , St Alphonsus Regional Medical Center, 1055 N Curtis, Boise ID 83706. (208) 378-3421.

September 21-23 in Minneapolis, Minnesota. The MSRC announces its 25th Annual Convention at the Radisson South & Plaza Tower. Topics include all categories of adult and pediatric respiratory care, focusing on the changing health-care system as it relates to the profession. Approximately 70 vendors exhibit the latest advances in equipment and technology. Events include a golf tournament and welcome party, as well as sputum bowl finals, volleyball, and roller-blading at the Hubert H Humphrey Metrodome. Contact Steven Due at (612) 863-6890.

September 22-23 in Napa, California. Chapter 10 of the CSRC, the American Lung Association of the Redwood Empire, and the Respiratory Therapy Program at Napa Valley College present the 12th Annual Napa Valley Conference, "Current Concepts in Cardiopulmonary Care." Contact Kate Benscoter at (707) 253-3145.

September 24-27 in San Juan, Puerto Rico. The Puerto Rico Society for Respiratory Care (PRSRC) hosts the Third Latin American Congress for Respiratory Care at the San Juan Convention Center. Featured presentations cover all aspects of critical care and pulmonary rehabilitation. Contact PRSRC President Norma Cruz-Hiraldo at (809) 764-5788.

September 27-28 in Honolulu, Hawaii. The HSRC announces its 21st Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Contact Helen Ono RRT, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532, fax (808) 547-9535.

September 28-29 in Albany, New York. The Northeastern Chapter of the NYSSRC presents its Annual Teaching Day at the Desmond Inn. Featured topics include the hantavirus pulmonary syndrome, current usage of hyperbaric medicine, and permissive hypercapnia. Other activities include sputum bowl competitions and an open forum panel with area pulmonologists. Contact Dawn Bentley or Bill Wenzel, Respiratory Care Department, St Peter's Hospital, 315 S Manning Blvd, Albany NY 12208. (518) 454-1062 or (518) 454-1066.

October 14 in Colorado Springs, Colorado. The Southern Chapter of the CSRC presents the First Annual Southern Colorado Pulmonary Medicine Symposium at the Hilton Inn. Presenters include Neil MacIntyre MD, Keith Meredith MD, Tom Petty MD, Bruce Rubin MD, and Jack Wanger MBA RRT RPFT. Contact Barry Beard at (719) 630-5211 or fax (719) 577-2535.

October 26-27 in Sturbridge, Massachusetts. The Sturbridge Host Hotel and Conference Center is the site of the 17th Annual Meeting of the MSRC. Featured topics include PEP therapy, health care reform, and air medical transport. Social events include a sputum bowl, golf tournament, and dancing. Contact Gina Farquharson, MSRC Executive Office, 945 Concord St, Framingham MA 01701. (508) 620-4505.

February 14-17, 1995 in Reno, Nevada. The NSRC and the American Lung Association of Nevada present the 14th Annual High Sierra Critical Care Conference at the

Peppermill Hotel/Casino. The four-day conference covers critical care topics in adult, pediatric, and neonatal medicine. Contact Colleen Banghart, American Lung Association of Nevada, PO Box 7056, Reno NV 89510.

OTHER MEETINGS

September 15-16 in Midland, Texas. The cardiopulmonary department of Memorial Hospital in Midland TX and the University of Texas Health Science Center at San Antonio present their CardioPulmonary Conference. Featured topics include critical care in the Year 2000, the scientific and political history of respiratory care, worldwide evolution of the brain-death concept, and foregoing life-sustaining therapy vs euthanasia. 12 hours of CRCE credit are available. Contact Helen Strange at (915) 685-1655, (915) 685-1623, or fax (915) 685-4979.

September 16-18 in Kansas City, Missouri. The Missouri/Kansas Association of Cardiovascular and Pulmonary Rehabilitation announces its Seventh Annual Meeting, "Steps to a Successful Future," at the Hyatt Regency Crown Center. Presentation topics include sleep apnea, cardiopulmonary rehabilitation, managed competition, and patient outcomes. Contact Mary Cullen CRTT, Pulmonary Rehabilitation Coordinator, Menorah Medical Center, 4949 Rockhill Rd, Kansas City MO 64110. (816) 276-8711.

September 26-27 in Ann Arbor, Michigan. The University of Michigan Medical School sponsors "Update on Pulmonary and Critical Care Medicine" at the University's Towsley Center. The course focuses on interstitial and inflammatory lung disorders, evolving technologies, and recent concepts in pulmonary medicine, critical care, and infectious diseases. Approved for 14 hours of AMA Category 1 credit of the American Medical Association's Physicians Recognition Award program. Write to Registrar, Towsley Center for Continuing Medical Education, Department of Postgraduate Medicine and Health Care Professions, University of Michigan Medical School, PO Box 1157, Ann Arbor MI 48106-1157.

September 29-October 2 in London, Ontario, Canada. The Respiratory Therapy Society of Ontario (RTSO) presents its 22nd Annual Education Forum at the Radisson Hotel. Contact the RTSO Head Office, 122 Cumberland St, Suite 200, Toronto, Ontario, Canada M5R 1A6. (800) 267-2687.

September 29-October 2 in Dearborn, Michigan. The Extracorporeal Life Support Organization (ELSO) announces its Sixth Annual Meeting at the Ritz Carlton Hotel. Contact the ELSO Office at (313) 998-6600.

February 5-8, 1995 in Breckenridge, Colorado. The American College of Chest Physicians sponsors "Cardiopulmonary Wellness and Rehabilitation" at The Village at Breckenridge Resort. Contact Arlene Karavich, American College of Chest Physicians, 3300 Dundee Rd, Northbrook IL 60062-2348. (708) 498-1400.

March 12-15, 1995 in Denver, Colorado. The AARC and the National Jewish Center for Immunology and Respiratory Medicine announce the 5th International Conference on Pulmonary Rehabilitation and Home Ventilation at the Hyatt Regency Hotel. Postgraduate workshops on pulmonary rehabilitation, home ventilator care, sleep disorders, smoking cessation, and transtracheal oxygen are offered before and after the conference. The deadline for receipt of abstracts for poster/oral presentations on original research and clinical observations is November 1, 1994. Contact the National Jewish Office of Professional Education at (303) 398-1000.

AARC
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Videotapes...

The Latest In Technology And Techniques For Respiratory Care Practitioners

Pulmonary Rehabilitation
By John E. Hodgkin, MD. Provides an overview of the sequence for pulmonary rehabilitation. Learn candidate evaluation and selection, rehabilitation team establishment, identification of short- and long-term goals, program components, assessment of patients' progress, and long-term follow-up. Discusses aerosol therapy, IPPB, oxygen therapy, and chest physiotherapy in the treatment of COPD. 60 minutes.
Item VT7

Sleep Apnea
By Alan K. Pierce, MD. Explains how sleep stages are related to respiratory patterns and blood gas values in both normal and abnormal subjects. Includes a discussion of the criteria for defining the sleep apnea syndrome and the distinguishing features of central, obstructive, and mixed causes of apnea. Also addressed is the efficacy of medical treatment to correct specific types of sleep apnea. 60 minutes.
Item VT11

Practical Management of ARDS
By David J. Pierson, MD. Adult respiratory distress syndrome is defined in this informative tape and its clinical features described, including incidence of risk factors and clinical predictors. Also extensively discussed is the use of PEEP to treat ARDS, including goals, complications, best or optimal PEEP levels, PEEP trials, and PEEP withdrawal, as well as general treatment, prognosis, and sequelae of ARDS. 60 minutes.
Item VT16

Pressure Support Ventilation
By Neil MacIntyre, MD. This presentation defines and describes the physiologial considerations attributed to PSV and the clinical situations when PSV may be useful. Includes comparisons when low-pressure levels and high-pressure levels of PSV are offered. Discussion includes using PSV to help overcome resistance for intubated patients and as an augmented ventilatory mode of weaning. 60 minutes.
Item VT17

Drainage of the Pleural Space: Management of Chest Tubes and Bronchopleural Air Leak
By Martha L. Tyler, RN, RRT. Learn the physiologic effects of abnormal pleural space function and the potential problems associated with chest tubes stripping, difficulties of bronchopleural air leaks with mechanical ventilation, the therapeutic goals of chest tube placement, and techniques for maintaining gas exchange with air leaks. 60 minutes.
Item VT21

Clinical Use of the Swan-Ganz Catheter
By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter placement and the situations in which SGC placement are useful are described in this video. The clinical value of the clinical variables monitored by SGC and the complications of its placement are detailed, including "damping" of the waveform, "overwedging," and optimal lung zone placement. 60 minutes.
Item VT22

Fetal Lung Development
By Charles Rosenfeld, MD. Examines the four anatomical phases of fetal lung development along with a discussion of the biochemical development of the fetal lung through gestation. Includes a description of the substances in surfactant, the importance of their timely development, and the methods used to assess fetus survivability by using tracheal aspirant to identify the necessary ratios of the phospholipids making up surfactant. 60 minutes.
Item VT23

Theory and Application of Neonatal Ventilation
By Robert Chatburn, RRT. Knob-turning in the neonatal intensive care unit should be a profound activity because it often has profound consequences. Adjustment of a single control on a ventilator generally has multiple effects, and thorough consideration of how controls are interrelated is essential for optimum care. Presents a well-organized and systemic approach for managing mechanical ventilation. 60 minutes.
Item VT25

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Tissue Oxygen Deiivery
By David R. Dantzker, MD. Discusses the relationship between adequate tissue oxygenation and adequate energy production, the development of lactic acidosis, and the probable role of tissue hypoxia in the multiple-organ failure of ARDS. Also discusses the factors that govern oxygen transport to the tissues and the variables that reflect the adequacy of tissue oxygen transport. 60 minutes.
Item VT26

Managing the Work of Breathing During Mechanical Ventilation
By John Marini, MD. Learn about the work of breathing and ways in which it can be minimized in the clinical setting on patients receiving mechanical ventilation. Focuses on diminishing the breathing workload through quantifying the effort during mechanical ventilation via direct measures such as oxygen consumption, electromyography, pressure-time index, and external work of breathing, as well as indirect measures. 60 minutes.
Item VT27

Pressure Support Update
By Neil MacIntyre, MD. Reviews pressure support ventilation and how it is used. Explains waveforms for airway pressure, flow, and volume. Also illustrates the measurement of inspiratory muscle loads by pressure-time index and muscle V_{O_2} and demonstrates how muscle efficiency changes as the character of work changes. Includes examples of low- and high-level PSV for elimination of the imposed work of breathing and as a weaning tool. 60 minutes.
Item VT28

The Hospitalized COPD Patient: 10 Commandments for the Clinician
By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. Details the 10 rules for the clinician to follow when the COPD patient enters the hospital. Emphasis is on the "what not to do." 60 minutes.
Item VT29

Monitoring Respiratory Mechanics During Mechanical Ventilation
By Robert L. Chatburn, RRT. Explains how physical and mathematical models are developed and applied and how they are incorporated into ventilator design to provide estimates of mechanics. Also explains some of the problems that can develop because of the limitations of the models. 60 minutes.
Item VT30

ARDS Review
By Tony Dal Nogare, MD. Discusses the latest developments in risk factors and treatment. Also covers the five diagnostic criteria that must be present to make an accurate diagnosis of ARDS, including clinical, radiographic, and physiologic criteria. 60 minutes.
Item VT31

Sleep Disorders
By Brian Foresman, DO. A discussion of the physiology of sleep and the kinds of respiratory and nonrespiratory sleep disorders seen in the hospital. Discusses how to spot sleep apnea, the problems caused by the inpatient hospital setting, sleep disorder diagnosis, and treatment. 60 minutes.
Item VT32

Uses and Abuses of Noninvasive Monitors in Respiratory Care
By Dean Hess, MEd, RRT. Presents a discussion of how much noninvasive monitoring is needed, proof of false positives, its financial impact, and its future. Discusses the various methods: transcutaneous, pulse oximetry, and capnography. 54 minutes.
Item VT33

Nutrition and Respiratory Care
By Rich Branson, RRT. Explains the relationships and interactions of malnutrition on ventilatory drive, respiratory muscles, lung structure, and immunity. Further discusses the effects of nutrients on the respiratory system, particularly the nutritional support needed by mechanically ventilated and COPD patients. 60 minutes.
Item VT34

Smoking Cessation: Intervention Techniques for the Respiratory Care Practitioner
By Kathleen A. Smalky, MD, MPH. Covers four major aspects of smoking cessation — the impact of smoking on illness and mortality, behavioral components, current cessation programs, and effective intervention. Discusses identification of the physically addicted smoker and the effects of nicotine withdrawal. 43 minutes.
Item VT35

Hospital Acquired Pneumonia
By Galen B. Toews, MD. Provides information on the epidemiology, pathogenesis, and management of nosocomial pulmonary infections. 52 minutes.
Item VT36

New Approaches to the Treatment of Asthma
By Roger Bone, MD. This videotape discusses physician-directed adult patient education and self-management programs using a peak flow meter. Includes information on the morbidity and mortality of asthma, therapy goals from the National Asthma Education Program, and the beta-agonist controversy. Explains the "step care" treatment approach and the complications of mechanical ventilation in status asthmaticus. 60 minutes.
Item VT37

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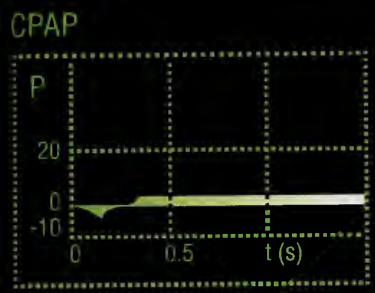

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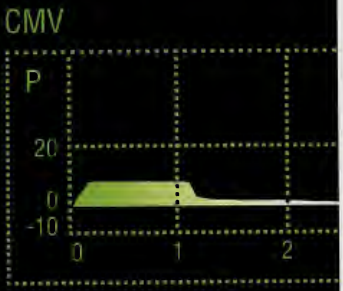

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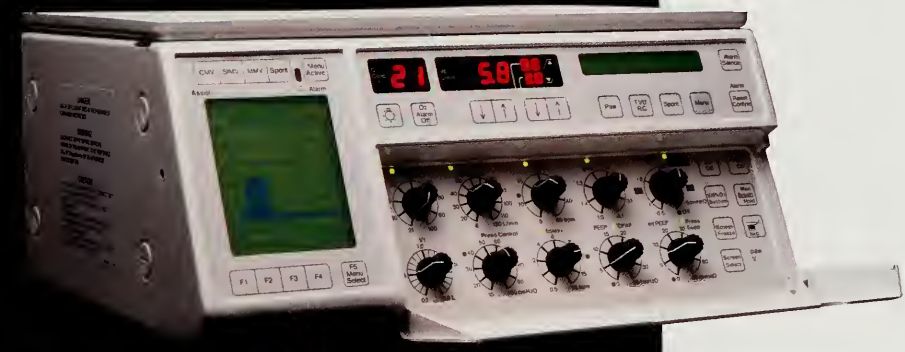

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